“COMPARATIVE STUDY OF THE CUFF PRESSURE BETWEEN AIR AND ALKALINIZED LIGNOCAINE IN GENERAL ANAESTHESIA”

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BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled **"COMPARATIVE STUDY OF THE CUFF PRESSURE BETWEEN AIR AND ALKALINIZED LIGNOCAINE IN GENERAL ANAESTHESIA"** is a bonafide record work done by **Dr. H.SAFEEBA BURVEEN,** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for MD, Branch X–Anaesthesiology

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DECLARATION

I **Dr. H.SAFEEBA BURVEEN,** solemnly declare that this dissertation entitled **"COMPARATIVE STUDY OF THE CUFF PRESSURE BETWEEN AIR AND ALKALINIZED LIGNOCAINE IN GENERAL ANAESTHESIA"** has been done by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, or diploma to any other University or board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Doctor of Medicine degree Branch X – Anesthesiology to be held in April 2013.

Place: Madurai **Dr. H.SAFEEBA BURVEEN**

Date:

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INTRODUCTION

Endotracheal tube was first introduced in mid 20th century .Till that time endotracheal tube was packed on the side of sub glottis by anesthetic swab for preventing gas leakage and ribbon guaze was tied on other end for removal of the tube at extubation. Endotracheal tube cuffs maintains the airway pressure during inspiration when the patient is on mechanical ventilation and prevents aspiration. Pressure generated by endotracheal tube cuffs usually transmitted to tracheal mucosa when elevated above 30 cmH2O causes ischemia of tracheal mucosal vessels which leads to ciliary loss, inflammation, ulceration, hemorrhage acutely, later on by tracheal stenosis and trachea esophageal fistula. The occurence of tracheal ischemia is directly related to the cuff pressure and duration of exposure.

Nitrous oxide, routinely used anesthetic gas readily diffuses inside the cuff thereby increasing the cuff pressure. Hyperinflation of the cuff causes tracheal mucosal lesions leading to sore throat, coughing, hoarseness which results in discomfort after extubation.

Tracheal intubation can cause airway changes, injury, and nerve damage. The most common complication that occurs after tracheal intubation is sore throat which is related to tracheal tube size and cuff design. Nerve damage caused by intubation is due to high intracuff pressure.

Apart from cuff pressure, techniques of endotracheal tube intubation plays an important role in airway trauma and postoperative sore throat. Rapidly adapting stretch receptors present over the tracheal mucosa is responsible for endotracheal tube induced cough. These receptors are highly sensitive to mechanical stimuli. Intubation with endotracheal tube and hyperinflation of the cuff causes coughing after extubation. Injection of lignocaine into endotracheal tube cuff causes its diffusion through the semipermeable membrane of cuff which anaesthetize the tracheal mucosa. This increases the airway tolerance during general anaesthesia, minimizes the hemodynamic changes and incidence of coughing after extubation.

Sodium bicarbonate increases the un-ionized form of local anesthetics which causes increase in diffusion of it through the cuff thereby reducing the dosage of local anesthetics.

AIM OF THE STUDY

PRIMARY AIM OF THE STUDY:

To compare the cuff pressure when inflated with air or alkalinized lignocaine during general anaesthesia.

SECONDARY AIM OF THE STUDY:

i. To evaluate the haemodynamic changes during the intraoperative period

ii. To evaluate airway tolerance during extubation.

iii. To evaluate the incidence of post-extubation tracheal morbidity in the form of

   a. Sore throat.

   b. Coughing.

   c. Hoarseness of voice.

HISTORY

Friedrich Trendelendurg -1871-described small, low volume inflatable cuffed rubber tracheal tube inserted into tracheostomy tube.

William McEwen -July 5, 1878-did oral intubation blindly for anaesthesia and airway obstruction.

Chevalier Jackson-1913- was the first person to use direct laryngoscopy for intubation.

Eisenmenger-1893- was the first person to describe about the tracheal tube with high volume inflatable cuff. In 1952-cuffed tracheal tubes were used during polio epidemic (before that non-cuffed was used)

.**Ivan.W.Magill & Edagar S.Rowbotham** –first used endotracheal tube for Facio-maxillary surgery.

Magill tubes-made of red mineralized rubber fits the airway.

Arthur Guedel &Ralph .M.Waters-1928-promotes cuffed endotracheal tubes.

Disposable plastic tubes replaces Red rubber tube for following reasons.

1. cross infection & release of toxins.
2. high pressure cuff causes tracheal erosion.
3. rubber tubes deteriorates on repeated autoclaving

Humphry Davy- was the first person to note the analgesic efficacy of nitrous oxide and he named it as "**Laughing gas".**

Horace Wells-used Nitrous oxide for tooth extraction.

Joseph Priestley- 1772- a French chemist who first synthesized Nitrous oxide, called it as "**phlogisticated nitrous air".**

Gardner Q.Colton-promoted the continued use of nitrous oxide.

Edmund Andrews-described the idea of using N2O with oxygen.

Nils Lofgren-1943-synthesised first amino amide ester Xylocaine (Lignocaine)

Bendt Lundquist was the first person who made experimental injection of lignocaine on himself

In 1949**-lignocaine** was first marketed.

PHARMACOLOGY OF LIGNOCAINE

Lignocaine is a synthetic amino amide derivative of local anesthetics with intermediate potency and duration. It is the standard local anesthetics to which all other agents are compared. It is the most commonly used local anesthetic. It can also be used as an antiarrhythmic. It can be given by all routes.(intravenous, intrathecal, infiltration, local applications like ointment, sprays, jelly etc.)

MECHANISM OF ACTION:

Lignocaine after entering the cell membrane binds to the inner portion of the resting sodium channel receptor. It prevents the propagation of nerve impulse through the ion-selective sodium channel in the nerve membranes. This decreases the depolarization rate, so that the threshold potential is not reached. Thereby, action potential generation is inhibited, without altering the membrane potential.

PHYSIOCHEMICAL PROPERTIES:

Molecular weight : 234

Weak base with a pka : 7.6 – 7.8

Stable, not decomposed by heating, acids or alkalies

Compared to bupivacaine, it is less lipid soluble

PHARMACOKINETICS:

Absorption:

Lignocaine is absorbed into the blood stream from the site of application or injection. Blood flow to that area and use of epinephrine determines the rate of absorption.

Metabolism and Excretion:

It is metabolized in liver by oxidative de-alkylation to mono ethyl glycinexylidide which on further hydrolysis produces xylidide. Hepatic blood flow determines its metabolism. 80% activity of the parent drug is by Mono ethylglycine xylidideand 10% of its activity by xylidide. 75% of xylidide is excreted in the urine as 4 – hydroxyl – 2,6 – dimethylaniline.

Onset of action:

The onset of action varies with the site of application of lignocaine. For topical anaesthesia its onset - 5-10 minutes, For conduction anaesthesia in small nerves- 5-10 minutes For large nerves - 10-15 minutes For Intravenous administration -1-2 minutes. 70% of the drug binds to α1 acid glycoproteins.

Volume of distribution 91 litres.

Lignocaine has a triphasic distribution .

Rapid distribution phase (α): during this phase, the drug is distributed to highly vascular regions with t ½ α of 1 minute.

Slow disappearance phase (β): here the drug is distributed to slowly equilibrating tissues with t ½ β of 9.6min.

Finally the drug goes to slow transformation and

Excretion phase (δ) : Where the t½ δ is 1.6 hrs.

Clearance - 0.95 litres per minute.

Availability:

- 2% lignocaine ointment, 5% lignocaine ointment
- 10% lignocaine spray
- 2% lignocaine Jelly
- 4% lignocaine aqueous solution
- 4% lignocaine viscous
- 4% lignocaine with adrenaline as 1 in 200000 dilution – 30 ml vial.
- 2% lignocaine with adrenaline as 1 in 200000 dilution

- 2% lignocaine – plain – 30 ml vial –contains methyl and propyl paraben as preservative
- 2% lignocaine (xylocard) without preservative – 50 ml vial for intravenous use
- 5% heavy 2 ml ampoules which contain 50 mg of lignocaine / ml with 75 mg – 100 mg of dextrose

PHARMACODYNAMICS:

Local actions:

It acts locally causing loss of pain, touch and temperature sensation. Motor power and vasomotor tone is also lost in the region supplied by the nerves blocked.

SYSTEMIC ACTIONS:

Its action depends on the site / route (intravenous) of administration.

Cardiovascular system:

It stabilizes the myocardial cell membrane. It depresses myocardial automaticity by inhibiting the action potential and reducing the duration of effective refractory period. At higher concentration, cardiac conductivity and contractility are depressed. All these effects result from direct cardiac muscle membrane changes occurring due to cardiac sodium channel blockade. It suppresses ectopic foci by stabilizing the membrane of damaged and excitable cells.

Vascular smooth muscle :

It acts on vascular smooth muscle producing vasodilatation.

Respiratory system :

Lignocaine depresses the ventilatory response to low PaO2 (hypoxic drive).Direct exposure of local anaesthetic to higher centres like medulla or peripheral nerves (intercostal ,phrenic nerve) causes respiratory depression. - Since it relaxes bronchial smooth muscle, Intravenous lignocaine may be effective in blocking the reflex bronchoconstriction associated with intubation.

Central nervous system :

Lignocaine causes a sequence of stimulation followed by depression. It causes sedation on intravenous administration. Intravenous administration

decreases cerebral blood flow and attenuates the rise in intracranial pressure that accompanies intubation. Lignocaine injection is capable of reducing the Minimal Alveolar Concentration of volatile anaesthetics by 40%.

Musculoskeletal:

Lignocaine causes lytic degeneration, edema and necrosis of muscle fibres.

Hematological :

It enhances fibrinolysis thereby increasing the clotting time.

Indications:

For infiltration block, peripheral nerve blocks, topical anaesthesia, intravenous regional anaesthesia, intra cuff injection (with sodium bicarbonate), spinal and epidural anaesthesia

1. **Antiarrhythmic:**

Lignocaine is a class IB anti arrhythmic drug. It is used in the treatment of

- Ventricular tacharrhythmias
- Arrhythmias due to acute myocardial Infarction during cardiac surgery
- Digitalis toxicity – since it does not worsen AtrioVentricular– block

2. It is used to prevent increases in intracranial pressure during intubation – antitussive effect may be the reason for this action.

3. It suppresses noxious reflexes (coughing) & sympathetic stimulations associated with endotracheal suctioning and intubation.
4. It is used intravenously as an analgesic for chronic pain states
5. It is used as a supplement to general anaetheisa.

Contraindications:

1. It should not be used in patients with (hypersensitivity) allergic disorder.
2. It should not be used along with vasoconstrictor in digits of hand, feet and penis.
3. It is contraindicated in patients with Stokes Adams syndrome, severe degree of heart block

Doses:

Maximum recommended dose :

Plain-3mg / kg

plain with adrenaline-7 mg / kg

plain without preservative for reflex suppression -1.5 mg / kg iv.

Drug interactions:

β Blockers: Co administration of beta blockers, increases the toxicity by increasing the serum levels of lignocaine by reducing lignocaine's metabolism.

Anticonvulsant agents: It increases the metabolism of lignocaine.

Non depolarizing muscle relaxants: lignocaine potentiates the blockade of non- depolarizing muscle relaxant.

Opioids and α 2 adrenergic agonists: These drugs increases the analgesic action of lignocaine.

Anti-arrhythmic agents: anti arrhythmic effect of lignocaine is increased by these agents.

Toxicity:

Lignocaine toxicity is due to systemic absorption of locally administered lignocaine or due to accidental intravenous administration of large doses of lignocaine. The central nervous system is most commonly affected.

Symptoms and blood levels:

Light headedness, tinnitus, circumoral and tongue numbness(anticonvulsant and antiarrhythmic activity): 4 micrograms / ml

visual disturbances: 6 micrograms / ml

muscular twitching: 8 micrograms / ml

convulsions : 10 micrograms / ml

unconsciousness : 12 micrograms / ml

coma: 15 micrograms / ml

respiratory arrest: 20 micrograms / ml

cardiovascular collapse: 26 micrograms / ml

Treatment of toxicity:

Lignocaine toxicity can be identified earlier by continuous monitoring of Cardiovascular and Respiratory status.

• Barbiturates or benzodiazepines are used to treat convulsion.

• Succinylcholine 1mg/kg used to paralyze and to intubate the patient to have airway control during seizures.

• Ventricular fibrillation can be treated by defibrillation.

• 100 % oxygenation, intubation and ventilation.

• Maintain Blood Pressure by rapid infusion of Intra Venous fluids, use of vasopressors and put the patient in Trendelenberg's position.

• Treat electrolyte imbalance.

Adverse effects:

1.Allergic and hypersensitivity reactions :

Methyl paraben used as a preservative causes allergic and hypersensitivity reactions.

2.Cardiovascular system : hypotension, bradycardia.

PHARMACOLOGY OF NITROUS OXIDE

Molecular Formula	:	N2O
Molecular Weight	:	44gm/mol
Appearance	:	colour less gas
Density	:	1.97 gm/L
Melting point	:	-90.86degree celcius
Boiling point	:	-88.48 degree celcius
Vapour pressure	:	5150 KPa
Critical temperature	:	36.5 degree celcius

Minimum alveolar concentration: 104

SYNTHESIS:

Nitrous Oxide is synthesized by heating ammonium nitrate by thermal decomposition.

BIOTRANSFORMATION:

It is mostly excreted through lungs, small amount through skin, <0.01% undergoes reductive metabolism in GIT by anaerobic bacteria.

Effects on organ system:

Cardiovascular system:

It causes direct myocardial depressant effect. It increases the catecholamine release and thereby increases the Blood Pressure, Pulse Rate, Cardiac output (indirectly).Nitrous oxide increases the incidence of epinephrine induced arrhythmias. It increases the pulmonary vascular resistance by constriction of pulmonary smooth muscle thereby increases the right ventricular end-diastolic pressure.

Respiratory System:

It increases the respiratory rate, but decreases the tidal volume. It depresses the hypoxic drive mediated by peripheral chemoreceptor (carotid bodies).

Central nervous system:

It increases the cerebral blood flow, cerebral metabolic oxygen demand (CMRO2),and also causes mild increase in intracranial pressure.**Renal system:**

It decreases the renal blood flow by increasing the renal vascular resistance, thereby decreases the GFR, urine output.

Hepatic system:

It decreases the hepatic blood flow.

Gastrointestinal system:

It activates the vomiting &chemoreceptor trigger zone in medulla and causes nausea &vomiting.

Neuromuscular system:

It Does not provide good muscle relaxation. It doesn't trigger malignant hyperthermia.

ADVERSE EFFECTS:

It diffuses through the air filled cavities thereby increases the intra cuff pressure which results in sore throat after extubation.

It inhibits methionine synthetase necessary for myelin formation & thymidylate synthetase necessary for DNA synthesis

On prolonged exposure causes megaloblastic anemia, pernicious anemia, and peripheral neuropathy.

TRACHEAL ANATOMY

The trachea is a membranocartilaginous tube. It extends from sixth cervical vertebra to the fifth thoracic vertebra. It bifurcates at lower border of fourth thoracic vertebra into two bronchi, the right and the left. The right bronchus is shorter, wider and in line with trachea ,whereas the left bronchus is longer and narrower. Compared to adult, children have shorter and mobile trachea which is more deeply placed posteriorly. It measures about 11 cm. in length; its diameter, from side to side, is from 2 to 2.5 cm., being always greater in the male than in the female. During inspiration, bifurcation gets lowered at C6 but during expiration it moves about one vertebral level. Trachea is kept patent by U-shaped ring of elastic cartilage embedded in its wall. Trachealis muscle which is made of smooth muscle fibres connects the posterior free ends of the cartilaginous ring.

Relations:

Anteriorly: sternum, thymus gland, arch of aorta, common carotid & Brachio-cephalic arteries, left brachiocephalic vein.

Posteriorly: oesophagus, left recurrent laryngeal nerve.

Right side: azygous vein, right vagus nerve, pleura.

Left side: arch of aorta, left common carotid artery, left subclavian artery, left Brachio-cephalic artery, left vagus nerve, left phrenic nerve, pleura

Blood supply:

Upper two third of the trachea is supplied by inferior thyroid artery and Lower one third of the trachea by bronchial artery

Nerve Supply:

Vagus and recurrent laryngeal nerve supplies the sensory innervation to the trachea .Trachealis muscle is supplied by sympathetic nerves.

Lymphatic Drainage:

Pretracheal, paratracheal, & deep cervical lymph nodes provides lymphatic drainage for the trachea.

TRACHEAL HISTOLOGY

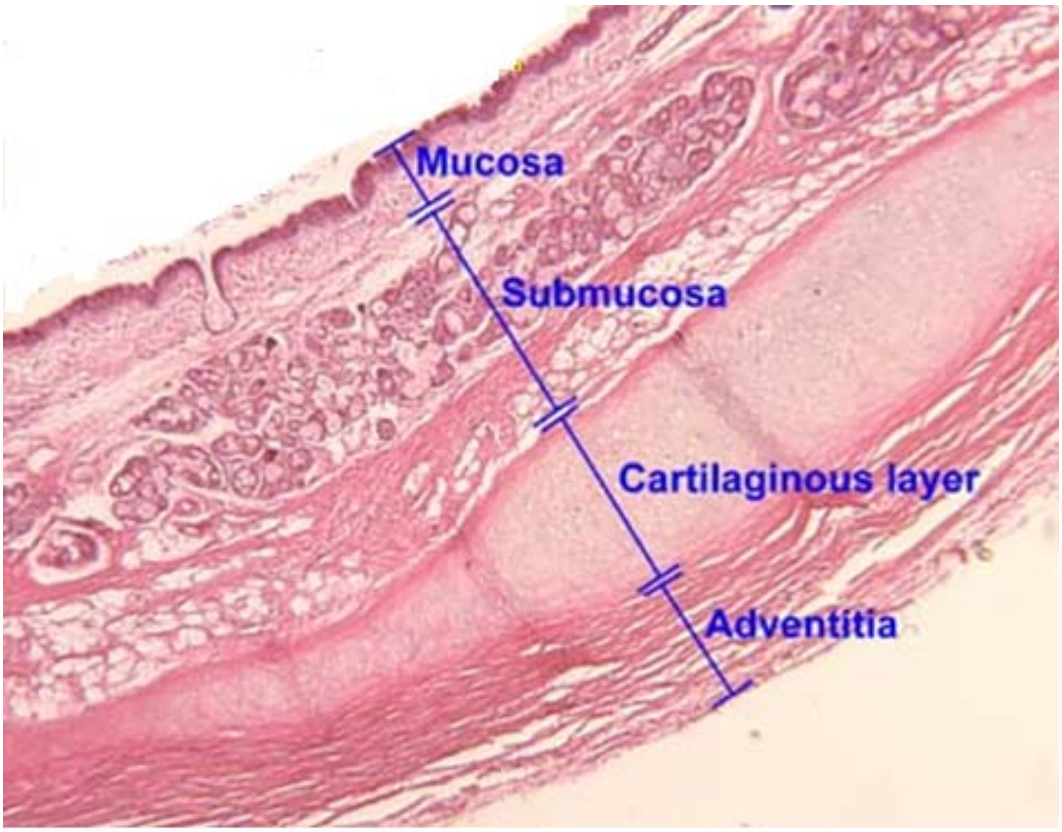
Tracheal mucosal epithelium-psuedostratified ciliated columnar epithelium (with numerous globlet cells) with thick basement membrane.

Lamina propria-loose connective tissue layer forms the mucosal layer along with epithelial layer.

Submucosal layer-made of longitudinal elastic fibres with serosanguinous glands.Outside this connective tissue layer is made of **hyaline cartilage** which prevents tracheal lumen from collapsing.

Muscularis propria-made of horizontally arranged muscle fibres that bridge the cartilage. It acts to adjust the tracheal diameter. Adventitial layer-outer most layer, made of connective tissue.

TRACHEAL HISTOLOGY:



TRACHEAL INTUBATION

Tracheal intubation –Introducing a flexible plastic tube into the trachea to keep airway patent and thereby facilitating mechanical ventilation and to prevent the occurrence of airway obstruction. The orotracheal intubation is the most common route of endotracheal intubation. Nasotracheal intubation can also be done. It is the invasive method in which general anaesthesia with neuromuscular blocking agents are used for intubation.

Intubation is facilitated by means of conventional laryngoscope, fibreoptic bronchoscope, video laryngoscope etc.to visualize the glottis. After intubation pilot balloon with cuff is inflated to keep endotracheal tube in place to prevent leakage of gases from the trachea and aspiration of gastric contents to the trachea.

INDICATIONS:

1.Unconcious patient.

2. Airway obstruction.

3. Hypoxaemia.

4. Diagnostic &Therapeutic manipulation of airway.

METHODS TO CONFIRM ENDOTRACHEAL INTUBATION:

Endotracheal intubation is confirmed by following ways:

1.Bilateral equal movement of the chest during ventilation. Bilateral equal air entry heard with help of stethoscope. Water vapour present over the lumen of the tube during expiration without gastric contents.

2. Direct visualization of passage of tube by means of laryngoscope

3. Capnography is also used to confirm the endotracheal tube position

4. Distal tip should be 2cm above the carina confirmed by chest X-ray.

5. Calorimetric endtidal CO2 detector, self inflating esophageal bulb, an esophageal detecting device etc are used to confirm endotracheal intubation.

COMPLICATIONS:

1.Sorethroat

2.Injury to airway (trauma to lips,gums etc)

3.Loosening,dislodgement of teeth

4.Arrhythmias,high blood pressure

5.Increased intracranial pressure, and intraocular pressure

6.Esophageal intubation

7.Laryngospasm,bronchospasm

CUFF SYSTEM, DESIGN & MATERIAL

Requirements for the proper design of endotracheal tube cuffs specified by The American Society of Testing And Materials (ASTM)

1.Maximum distance from the tip of the tube to the patient end varies with tube size

2. Edge of cuff should not encircle the murphy eye.

3.Cuff should inflates symmetrically without herniation over tube tip.

CUFF SYSTEM:

It includes

1. Cuff
2. An inflation system which consists of
   a. An inflation tube
   b. A pilot balloon
   c. An inflation valve

Main advantages of cuff are

- It centers the tube in trachea
- It prevents gas leakage along the tube during positive pressure ventilation.
- It prevents the entry of upper airway contents into

trachea by providing a seal between the tube and tracheal wall

MATERIALS:

Characteristics of ideal tracheal tube materials are as follows:

- It should be transparent
- It should be inexpensive
- It should be non-toxic
- It should have long term usage with frequent sterilization.
- It should not be inflammable
- It should be free of latex.
- Both inner & outer surfaces of the tube should be dry and smooth for introducing suction catheter etc. and to prevent trauma to airway.
- It should be non- kinkable
- Even tube made of thin wall should be of sufficient strength.
- It should not react with lubricants &anesthetic agents.
- It should be thermolabile according to patients body temperature.

No substance will meet all these characteristics.

TYPES OF ENDOTRACHEAL TUBE BASED ON MATERIAL:

1. Red rubber tube
2. Poly vinyl chloride (PVC) tube
3. Silicon tube

Red rubber tube:

It is still in use. It can be sterilized and used multiple times. Disadvantages: Lack of transparency, latex allergy, easily plugged by secretions are the disadvantages of red rubber tube.

Polyvinyl chloride (PVC) tube:

- Most widely used now-a-days for oral/ nasal intubation.
- Disposable, low cost, resistant to kinking.
- Radio-opaque marker along the tube.
- Thermoplastic –get stiffen/soften according to body temperature.
- Transparent –allows for observing movement of moisture during respiration & substance in the lumen.
- Smooth surface allows easy passage of suction catheter

Silicon tube:

- Rarely used
- Reusable after sterilization.
- Expensive

TUBE DESIGN:

- Outer and inner wall of the tube is circular so that less prone for kinking
- Proximal machine end can be shortened and distal end is bevel shape which faces left.
- Murphy eye opposite the bevel edge function as the path for gas flow when there is an obstruction in bevel end.
- Radio opaque line situated along whole length of the tube to confirm the tube position after intubation.

SIZE OF THE ENDOTRACHEAL TUBE (Internal diameter)

Male: 8 mm , Female: 7.5mm

New born upto 3 months: 3mm

3-9 months: 3.5mm

9-18 months: 4mm

2-6 years: AGE/3+ 3.5mm

>6 years: AGE/4+4.5mm

DEPTH OF ENDOTRACHEAL TUBE INSERTION:

ADULT : MALE: 23cms FEMALE: 21cms

CHILDREN:

Orotracheal intubation: AGE/2 +12cms

Nasotracheal intubation: AGE/2 +15cms

CUFF TYPES :

Low volume, high pressure cuff

High volume, low pressure cuff.

Low volume, High pressure cuff:

It has short diameter with small residual volume requires high pressure to seal with trachea.

Advantages:

It is most commonly used in adults, inexpensive, can be reused.

It has less incidence of sore throat and good protection against aspiration.

It does not obscure the view during intubation.

Disadvantages:

High pressure cuff are more prone to tracheal ischemic injury after prolonged usage. Therefore for prolonged procedure /elective ventilation it is better to use low pressure cuff.

High volume, low pressure cuff:

It has large diameter with large residual volume provides better sealing without tracheal stretching.

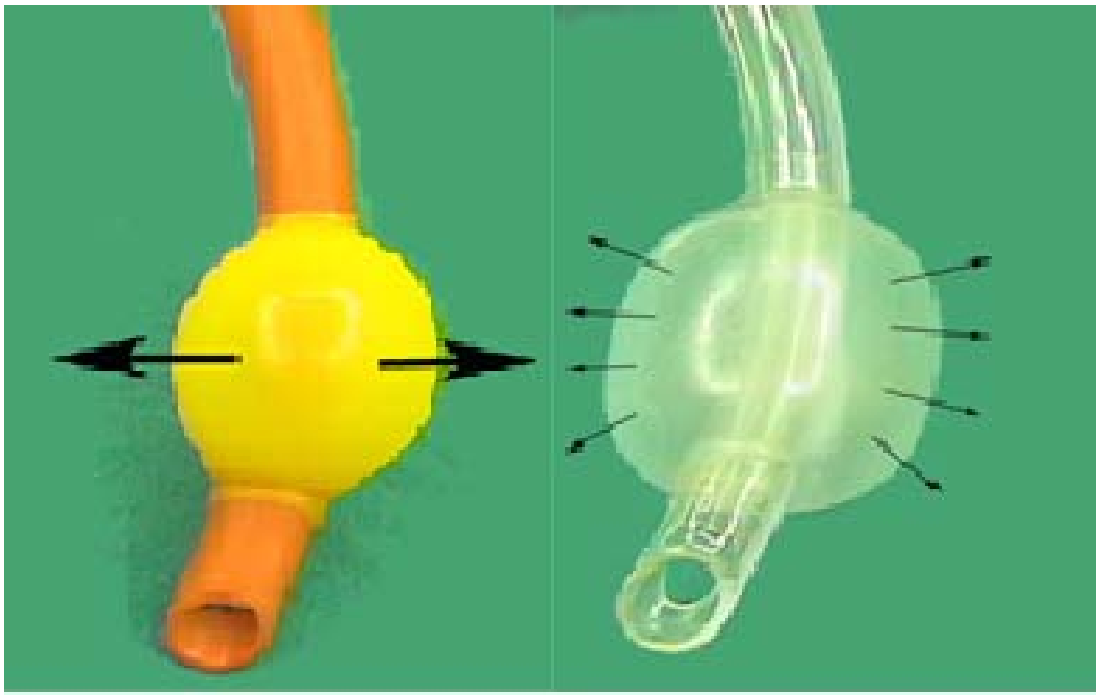
Advantages:

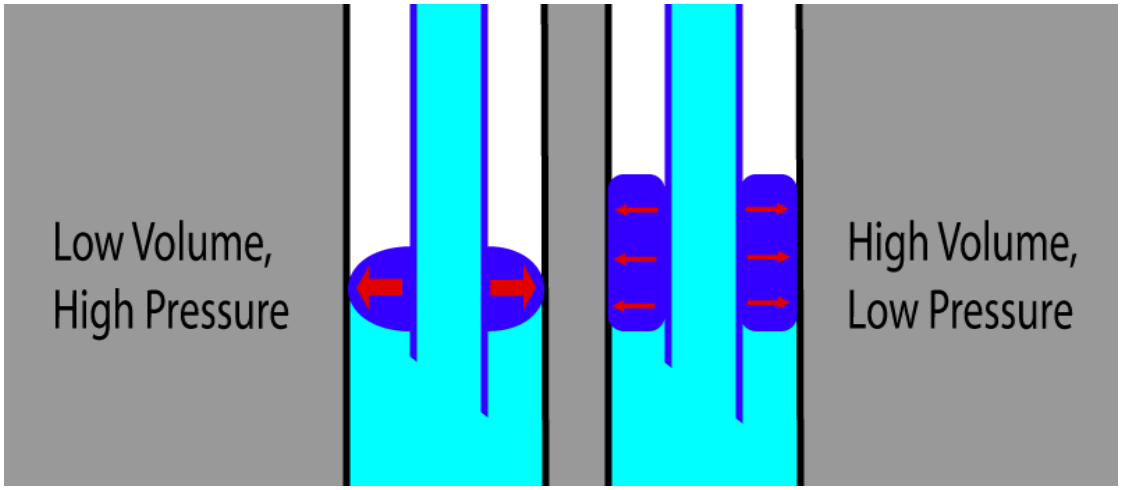
It provides intracuff pressure similar to pressure on the tracheal wall.It can be used for prolonged intubation.

Disadvantages:

1. There is a greater incidence of sore throat associated with low pressure cuff.
2. These tubes disturb the tube tip view, get torn easily and more prone for tube dislodgement.
3. Use of N2O diffuses into the cuff will increase the pressure over tracheal mucosa.

SCHEMATIC REPRESENTATION OF LOW VOLUME HIGH PRESSURE &HIGH VOLUME LOW PRESSURE CUFF





EFFECT OF NITROUS OXIDE ON CUFF PRESSURE

During administration of Nitrous Oxide, there is an increase in cuff pressure and volume when inflated with air. Increase in cuff pressure is directly related to

1. Nitrous Oxide partial pressure.
2. Endotracheal tube cuff wall permeability.
3. Duration of anaesthesia.

The most of the disposable tubes were made of polyvinyl chloride. These cuffs will reach recommended cuff pressure limits when exposed to nitrous oxide anaesthesia for a period of time. Tubes made of silicon & polyvinyl chloride are more susceptible to nitrous oxide diffusion. Endotracheal tube cuffs with thin wall is also responsible for early diffusion of nitrous oxide so that critical cuff pressure will reach within 60 mins.

MECHANISM OF NITROUS OXIDE DIFFUSION:

The walls of the closed gas (air) filled compartments are more compliant. Nitrogen present in the air filled cavities has low blood solubility with blood: gas partition coefficient of 0.015.Nitrous oxide can easily transfer across the membrane barrier in contrast to nitrogen so that nitrogen gets trapped inside the air filled cavities (Nitrogen is 35times less blood soluble when compared to nitrous oxide). During Nitrous oxide anaesthesia, there is a doubling of volume

of the cuff which depends on alveolar nitrous oxide concentration.

Higher the alveolar nitrous oxide concentration can cause increase in compartment volume.

Discontinuation of nitrous oxide decrease the pressure in the cuff. Increase in cuff pressure occur during high altitude, alteration in head position, Oxygen diffusion, head and neck surgeries, coughing, muscle tone changes. Cuff pressure decreases during hypothermic bypass.

TABLE:1 ideal intra- cuff pressure for adults using PVC tubes.

PRESSURE	cm water	mm Hg
Ideal	20-30	15-22
High	>40	30
Low	<20	15

Tracheal mucosal injury related to lateral wall pressure and the duration of contact of tube with tracheal mucosa.Tracheal capillary pressure shouldnot exceed 30 cm of water or 15mm of Hg.

CUFF PRESSURE MONITOR

1. **Aneroid manometer** –It is most commonly used cuff pressure monitor. It is precise and accurate but requires calibration, expensive and carries risk of infection. Pressure limiting valves act as a resorvior thereby keep the intra cuff pressure in the preset range.
2. **Lanz pressure**-regulating valve: This monitor maintains the Cuff pressure around 30cm of water. It cannot be used in children. In case of high airway pressure it does not form good seal.
3. **Brandt tube system:** It can be used intra operatively during general anaesthesia. While using Nitrous Oxide anaesthesia, intra cuff pressure is stabilised by a large pilot cuff reservoir and an internal pressure regulating valve.
4. **Pressure easy cuff controller:** This type of cuff pressure monitor is expensive. It should be set according to peak inspiratory pressure. It is an electronically operated device.

CUFF PRESSURE MONITOR

POST OPERATIVE SORE THROAT

Incidence varies from 30-50 %

ETIOLOGY:

1. Tracheal mucosal injury due to intubation and instrumentation.
2. Ischemia of mucosa due to reduced blood flow as a result of increased intracuff pressure.
3. Erosion of mucosa due to irritation of airway (intubation etc.,)
4. Drying of mucosa due to inhalational anesthetics.

All the above factors lead to inflammation of airway.

RISK FACTORS:

1. Female sex.
2. Duration of anesthesia.
3. Position during surgery.
4. Nasogastric tube usage.
5. Suctioning of oropharynx.
6. Agents like succinyl choline and anesthetic sprays
7. Increased intracuff pressure.

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The most of the disposable tubes were made of polyvinyl chloride. These cuffs will reach recommended cuff pressure limits when exposed to nitrous oxide anaesthesia for a period of time. Tubes made of silicon & polyvinyl chloride are more susceptible to nitrous oxide diffusion. Endotracheal tube cuffs with thin wall is also responsible for early diffusion of nitrous oxide so that critical cuff pressure will reach within 60 mins.

MECHANISM OF NITROUS OXIDE DIFFUSION:

The walls of the closed gas (air) filled compartments are more compliant. Nitrogen present in the air filled cavities has low blood solubility with blood: gas partition coefficient of 0.015.Nitrous oxide can easily transfer across the membrane barrier in contrast to nitrogen so that nitrogen gets trapped inside the air filled cavities (Nitrogen is 35times less blood soluble when compared to nitrous oxide). During Nitrous oxide anaesthesia, there is a doubling of volume

FACTORS AFFECTING POSTOPERATIVE SORETHROAT:

1.Size of the Endo tracheal tube.

2.Technique of insertion of Endotracheal tube.

3.Lubricant application.

4.Pressure inside the cuff.

5.Nitrous oxide use during general anaesthesia.

TREATMENT:

1. Steroids and Non Steroidal anti-inflammatory agents

2. Benzylamine hydrochloride.

3. Alkalinized Lidocaine.

REVIEW OF LITERATURE

1) **Comparative Study between Intracuff buffered lignocaine and saline or air in patients undergoing ophthalmic surgeries with chronic airway disease.**

V.Jegadeesh ,V.V.Jayachandran, I.M.Banulakshmi,

Department of anesthesiology, Sankara Nethralaya Vision research Foundation, Chennai, Tamil Nadu.

V.V.Jayachandran et al (2009) studied that intra cuff injection of buffered lignocaine produces smooth extubation as cough receptors are blocked by more diffusion of basic form of local anesthetics across the PVC wall of the cuff.75 patients with history of chronic smoking &recently treated upper respiratory infection were randomized into 3 groups(25 in each group).Group A(air),Group B (saline6ml),Group C(6ml of 2% lignocaine+0.5ml 7.5%sodiumbicarbonate).They found out that extubation was smooth with buffered lignocaine group compared to Air Group & saline group. Sore throat incidence was higher in Air group compared to others.

2) **The effect of intra cuff alkalinized 2% lignocaine on emergence coughing, sorethroat, hoarseness in smokers.**

Laís Helena Camacho Navarro; Rodrigo Moreira e Lima, Anesthesiologists

Navvro et al (2012), conducted a randomized double blinded study in which he compared two Groups .50 patients undergoing general anesthesia using nitrous oxide with controlled ventilation were included in the study (n=25). Group L received 2%lidocaine with 8.4% sodium bicarbonate or Group S received 0.9% saline. The cuff was inflated to achieves pressure that prevents air leakage during positive pressure ventilation. He concluded that Group L superior to Group S in preventing sorethroat and cough.

3) **Comparative Study between Alkalinized intra cuff lignocaine & gel lubrication – in prevention of emergence phenomena after Endotracheal intubation.**

J.P.Estebe, Service d' Anaesthesisa reanimation chirurgicale. British journal of anestheisia 2004

A study was conducted on diffusion of buffered lignocaine solution using high-volume and low pressure ETT cuff. They also have done a study in 40

patients , 20 in each group- Group G received buffered lignocaine-filled Endotracheal tube cuff with gel lubrication , Group W received buffered lidocaine-inflated cuff with water lubrication ,Group C gets an air-inflated cuff with Endotracheal tube lubrication with water . Gel lubrication group reduces the incidence of sore throat after 24 hrs of extubation compared to other groups

Cough and restlessness before tracheal extubation were decreased in patients with the alkalinized lignocaine-filled cuffs compared with the air-filled cuffs. After extubation, nausea, vomiting, dysphonia and hoarseness were greater for patients with air-filled cuffs compared with the lignocaine-filled cuffs. No significant difference between the groups were recorded in arterial blood pressure and heart rate. In vitro data suggests that lower the NaHCO3 injection volume, the greater the release of lignocaine across a low-pressure, high-volume cuff endotracheal tube.

4) Efficacy of cuff inflation media to prevent post intubation-related emergence phenomenon: air, saline and alkalinized lignocaine.

European journals of anesthesia 2002

Shroff et al conducted a study among 150 patients undergoing surgery under GA with Controlled ventilation. Patients were divided into 3 equal groups of air, saline and alkalinized lignocaine as inflating agent. The

amount of inflating agent used, pressure inside the cuff, duration of surgery, amount of inflating agent withdrawn from the cuff and complications such as tube intolerance, coughing and bucking on the tube, hoarseness, restlessness, sore throat, breathlessness and laryngospasm were recorded. After intubation , the pressure inside the cuff was higher in the air group at five mins, thirty mins, one hour and just before extubation. Air volume was increased in air group just before extubation compared with a decrease in volume in the other groups. Lignocaine group had decreased tube intolerance, hoarseness and sore throat

5) The incidence and severity of postoperative sore throat after the use of Air, lignocaine, Saline for inflating endotracheal tube cuff.

Porter NE, Sidou V

Variables which were standardized are endotracheal tube cuff design , size of endotracheal tube, technique of intubation ,laryngoscope blade, airway placement, suctioning, anesthetic technique. ASA status I, II, or III, female, adult patients who underwent gynecological procedures under general anesthesia were included. VAS score and McGill Pain Questionnaire were administered to the Seventy five patients at 2 intervals between 1 to 3 hours & 22 to 25 hours postoperatively, for assessment of

postoperative sore throat. Kruskal-Wallis test was used for analysis and it suggested that the postoperative sore throat incidence was statistically insignificant among the 3 groups.

6) The study of various application of lignocaine in prevention of postoperative Tracheal morbidity.

Journal of clinical anesthesia 2002

Soltani et al conducted a study in 204 American Society Of Anesthesiologists I and II patients posted for ophthalmic surgeries with general anesthesia. Patients were allocated into 6 groups

G 1- Lignocaine spray (10%) over ETT

G 2- Lignocaine spray (10%) over oropharynx

G 3- ETT lubricated with 2% Lignocaine jelly.

G 4- Intravenous Lignocaine.

G 5- Intracuff Lignocaine

G 6 – ETT lubricated with Normal Saline.

From the study he concluded that there was a decrease in incidence of cough in I.V and Intra cuff Lignocaine Group. There was an increased in incidence of sorethroat with ETT lubricated with Lignocaine jelly, saline, 10% lignocaine spray compared to i.v and intracuff Lignocaine.

7) Effect of Intra cuff Lignocaine on Coughing during Emergence and Postoperative Sore Throat

Sagheer as, mazhar iqbal, khawar ali.

Sagheer et al studied in 100 patients regarding cough during and after extubation and postoperative sore throat at one and 24 hour after anesthesia. The endotracheal tube cuff was filled with 4 % lignocaine in one group and with air in the other group. There was significantly less frequency of cough in the lignocaine group. The incidence of postoperative sore throat was also decreased at first and twenty four hour after anesthesia. Using lignocaine to inflate the endotracheal cuff decreases the frequency of cough at extubation and postoperative sore throat.

8) The Efficacy of the Intra cuff buffered Lignocaine in general anaesthesiaUsingNitrousOxide.

Shin JC, Kim KS, Kim YJ, Choi WJ, Koo MS. Korean journal of anesthesiology 2008.

Shin et al done a study in patients undergoing thyroid surgeries .These patients were divided into four groups-30 each

Group A alkalinized lignocaine (2ml)

Group B alkalinized lignocaine (4 ml)

Group C air (6ml)

Group L plain lignocaine

Significant decrease in incidence of postoperative sore throat after 24 hrs in alkalinized lignocaine group in comparative to air or lignocaine group but both alkalinized group was statistically insignificant. ETT tolerance was good with alkalinized lignocaine group .Cough during extubation ,voice change after extubation was less in alkalinized lignocaine group compared to other two. From the study he concluded that buffered lignocaine is considered as better adjuvant to intracuff air using nitrous oxide anesthesia.

9) Comparing three different lignocaine regimens to inflate endotracheal tube cuff in preventing coughing during recovery from general anesthesia Huang .C.J

Journal - acta anesthesiology 1998.

80 ASA Class I-II patients who underwent elective surgeries were randomly assigned into 4 groups. Endotracheal tube cuff was inflated with one of following solution: normal saline 6 ml (Group A), 4% lignocaine 6 ml at room temperature (Group B), 4%lignocaine 5 ml + 7%

sodium bicarbonate 1 ml at room temperature (Group C), and 4% lignocaine 5 ml + 7% sodium bicarbonate 1 ml warmed to 38 degrees C (Group D). Changes of vital signs as well as the times of coughing in the course of extubation and post-extubation complications were recorded. Statistically significant difference in coughing was noted in experimental groups (B,C,D)when compared to Group A. Sore throat incidence was higher in Group A. Hemodynamic changes were higher in experimental groups (B,C) before extubation. Finally Huang et al concluded that alkalinized and warmed lignocaine prestored in endotracheal tube cuff can greatly reduce ETT induced coughing and provide smooth emergence after general anesthesia

10) The efficacy of intracuff lignocaine and iv lignocaine in prevention of emergence phenonmenon :comparative study in neurosurgical patients

Journal of neurosurgery anesthesiology 2006

Venkatesan et al performed in 82 patients undergoing neurosurgical procedures 41 patients in each group.

Group 1 received intracuff lignocaine 4%

Group 2 received lignocaine 1.5%iv before extubation

After extubation ,vital parameter recorded every 1 minute for 5 minutes and compared with baseline parameters. From the study he concluded that both the drugs produce similar effects in decreasing cough after extubation

11) Intra cuff, topical, intravenous lignocaine – comparative study- for prevention of coughing during emergence in general anesthesia .

Zamora et al conducted a study in patients with an ASA physical status of I or II over 18 years of age scheduled for elective surgery lasting between 60 and 120 minutes under balanced anesthesia with orotracheal intubation. These patients were randomly allocated to one of 3 groups (intravenous lignocaine, topical lignocaine, or intra cuff lignocaine) or to the control group.

Percentage of coughing in three groups:

Control group: 65% presented coughing

Topical lignocaine group: 26.3%

Intra cuff group: 15.8%

Intravenous group: 16%

Intravenous lignocaine and intra cuff lignocaine significantly reduce the incidence of coughing during emergence from anesthesia.

12) ETT cuff filled with Lignocaine 10%: Serum lignocaine concentrations,Hemodynamic changes and effects on organ systems.

European journal of anesthesiology 2000

Altintas et al conducted a study by instilling 10% lignocaine in endotracheal tube Cuff. Serum concentrations of lignocaine were studied. 70 ASA Class I or II patients who underwent plastic surgery were allocated into two groups:

group L: ETT inflated with lignocaine 10%

group S:ETT inflated with saline

The peak cuff pressure and hemodynamic changes were less in group L than group S.Group L had decreased severity and incidence of sore throat. Plasma concentration of lignocaine did not go upto toxic levels at the end of extubation. Lignocaine was better in reduction of postoperative sore throat both in severity and incidence.

13) Endotracheal tube cuffs inflated with lignocaine – a mode of drug administration: a study both in Vivo and In vitro.

Gilles dollo .Anesthesiology – European Journal 2001

Gilles dollo et al done a in vitro study regarding the release of lignocaine in three different forms (base form, hydrochloride form,

alkalinised form).From this study he concluded that alkalinised form of lidocaine requirement is less compared to other two forms. Endotracheal tube tolerance air < hydrochloride form alkalinised form (decreased pain score) in this order.

MATERIALS AND METHODS

Study Type: Interventional

Study design:

Prospective, randomized, double blinded study.

Study population:

After getting ethical committee approval and informed consent 50 patients of both sexes(male/female) who underwent breast & abdominal surgeries by general anaesthesia using Nitrous oxide at GOVT. RAJAJI HOSPITAL**,** MADURAI, were taken up for study.

Groups

GROUP A: Inflated with air upto pressure of 20 cm of water.

GROUP L: Inflated with alkalinized lignocaine(2% lignocaine : sodium bicarbonate 19:1 up to pressure of 20 cm of water.

Inclusion Criteria:

- ASA physical status class I ,II and III
- Age between 18 and 65 years

Exclusion Criteria: Patients with

History of known allergic to local anaesthetics,

Tracheostomy,

Laryngeal disease/surgery,

H/O smoking,

Those requiring insertion of nasogastric tube,

Those requiring more than 1 attempt to achieve tracheal intubation .

Probability sampling:

50 lots were randomized (25 in each group) from the people who were willing to take part in the study. All the patients stand an equal chance of getting into any group. All the patients were aware of the study and informed consent was obtained.

Materials:

1. Endotracheal tube (made of polyvinyl chloride single use)
2. Cuff pressure monitor, cuff syringe
3. 2% lignocaine (without adrenaline) 30 ml vial
4. 7.4% sodium bicarbonate,

Monitors used:

1. Pulse oximeter (PR,SPO2)
2. Non-invasive blood pressure monitor.(NIBP)
3. End-tidal carbon dioxide.(ETCO2)
4. Cuff pressure monitor.

Methodology:

In the preoperative waiting room detailed history and physical examination was done. Basic investigations were collected. Baselines data like pulse rate, blood pressure, SPO2. Group A &Group L were explained about the procedures and postoperative follow up pattern. The VAS was explained as 0-10 cm scale reading and patient was asked to tell the number.

All Patients were pre medicated with Inj. Glycopyrolate 0.2 mg intramuscularly 45 minutes before surgery. Monitors were connected. Intravenous cannula secured and connected to i. v fluids. Patient were preoxygenated with 100% O2 for 3 minutes. Patient is induced with Inj. fentanyl 2 micrograms/kg ,Inj. propofol 2mg/Kg i.v, Inj. Atracurium 0.5 mg/kg and intubated with 7.5 mm(females), 8.5mm(males) endotracheal tube.

GROUP A:Inflated with air upto pressure of 20 cm of water.

GROUP L: Inflated with alkalinized lignocaine(2% lignocaine : sodium bi carbonate 19:1 up to pressure of 20 cm of water.

Bilateral air entry checked and connected to closed circuit. Patient is maintained with N2O : O2 2:2, Inj.fentanyl 1 micrograms/kg every 45 mins, inj. atracurium in titrated doses. Baseline cuff pressure before N2O administration is monitored. Cuff pressure is recorded every 30 minutes thereafter .At the end of surgery, cuff pressure, tube tolerance, volume of air/alkalinized lignocaine deflated during extubation were noted. After adequate attempts of respirations, patient is reversed with inj.Glycopyrolate 10 mics/kg, inj. neostigmine 40 mics/kg and extubated after deflation of the cuff.

INTRAOPERATIVELY following parameters were monitored.

1. PR,BP,SPO,.ETCO2,Cuff pressure was noted before nitrous oxide administration ,&every 30 minutes till the end of surgery.
2. Tube tolerance ,BP, PR, volume of air /lignocaine deflated were noted.

POSTOPERATIVELY following parameters were noted:

Coughing,sorethroat,hoarseness were monitored 30 minutes after surgery in postanaesthesia care unit & 24 hours later.

Pain was assessed using visual analogue scale.

DATA ANALYSIS

PROFILE OF CASES STUDIED

Group A: Endotracheal tube filled with air

Group L: Alkalinised Lignocaine

Statistical Tools (To be included at the end of Materials and Methods)

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's chi square test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship

OBSERVATION AND RESULTS

Table 1: Age distribution

Age group	Group A		Group L	
	No	%	No	%
20-29 years	8	32	-	-
30-39 years	2	8	5	20
40-49 years	9	36	8	32
50 years & above	6	24	12	48
Total	25	100	25	100
Range	20-60		23-60	
Mean	39.7		40.3	
SD	12.8		10.5	
'p'	**0.8765 Not significant**			

With respect to age, both groups were comparable

AGE DISTRIBUTION

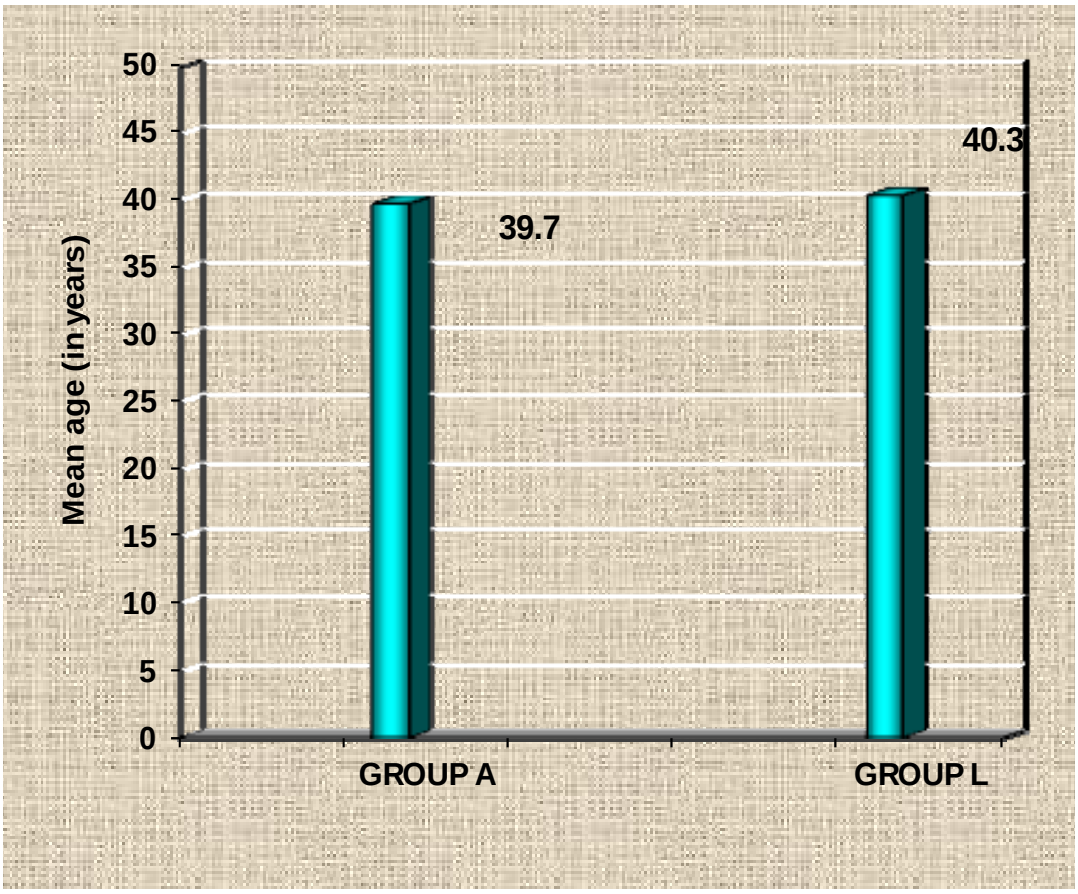

Table 2: Sex distribution

Sex	Group A		Group L	
	No	**%**	**No**	**%**
Male	12	48	9	36
Female	13	52	16	64
'p'	**0.5666 Not significant**			

Sex distribution shows no statistical significant difference.

SEX DISTRIBUTION

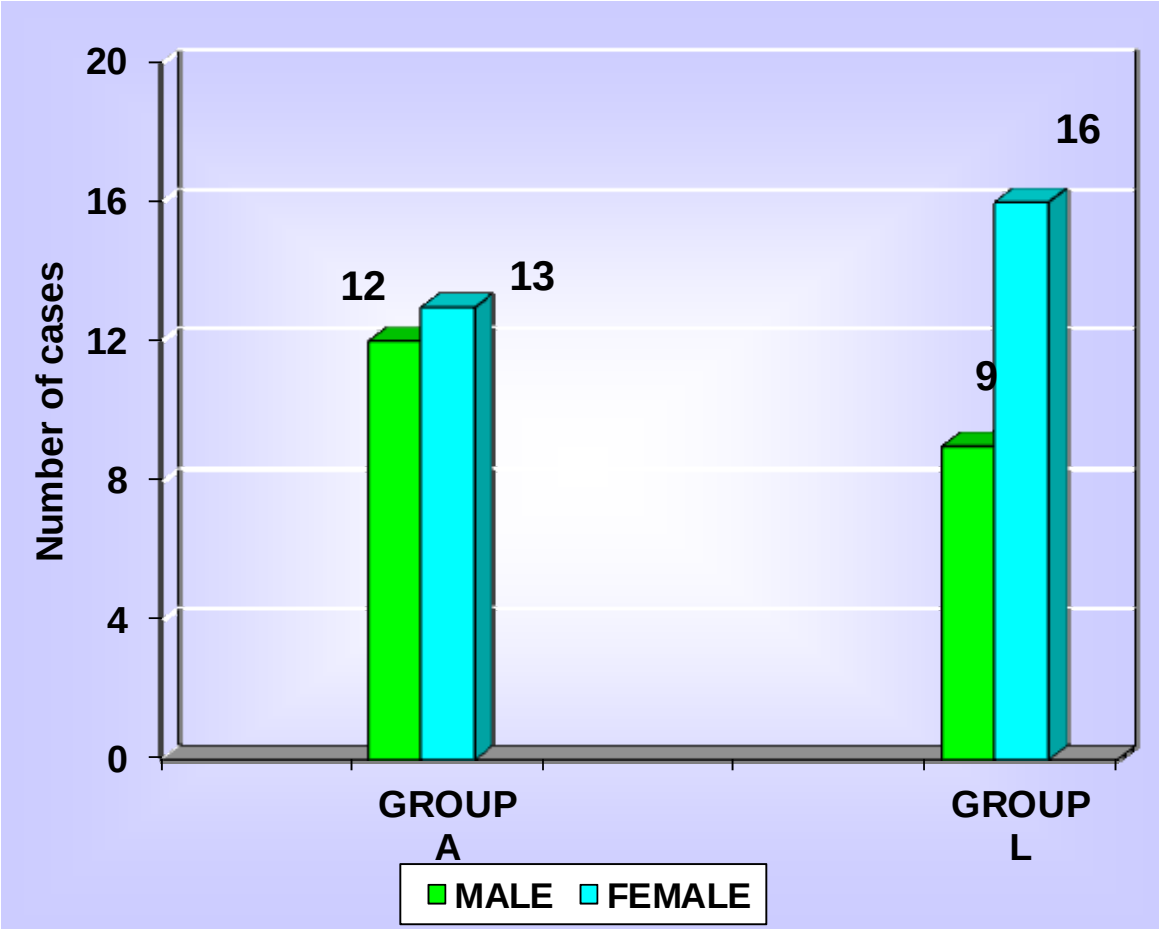

Table 3: ASA

ASA	Group A		Group L	
	No	**%**	**No**	**%**
I	12	48	6	24
II	10	40	19	76
III	3	12	-	-
Total	**25**	**100**	**25**	**100**

ASA risk is comparable in both groups.

ASA

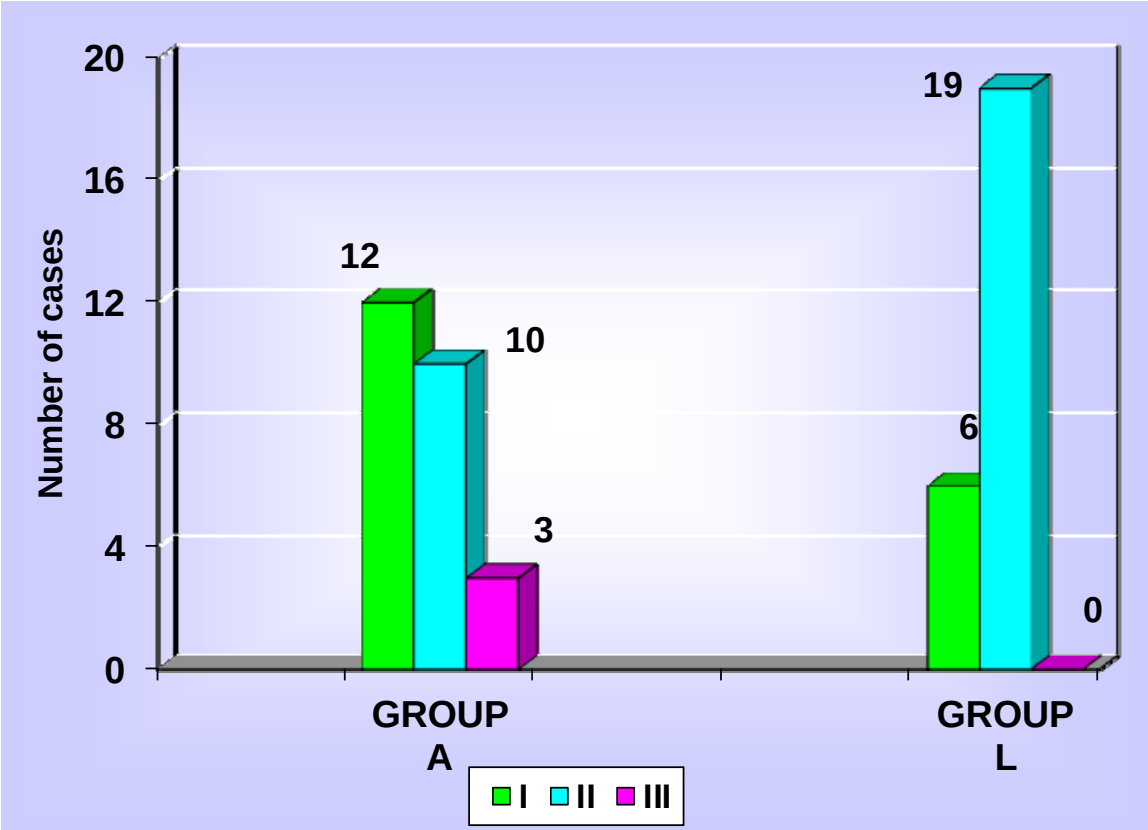

Table 4: Changes in Systolic Blood pressure

SBP at	SBP (mm/Hg)in				'p'	Significance
	Group A		Group B			
	Mean	SD	Mean	SD		
Before N2O	132	10.4	124.7	12.5	0.0783	Not significant
30 minutes	124.4	11.3	127.6	11.7	0.2982	Not significant
60 minutes	126.7	12.2	125.0	10.2	0.9922	Not significant
90 minutes	126.8	12.0	125.5	5.7	0.381	Not significant
120 minutes	125.4	10.1	124.9	5.4	0.559	Not significant
End of surgery	128.9	10.2	123.8	6.8	0.1724	Not significant

Systolic Blood pressure shows no statistical significant difference.

CHANGES IN SYSTOLIC B.P.

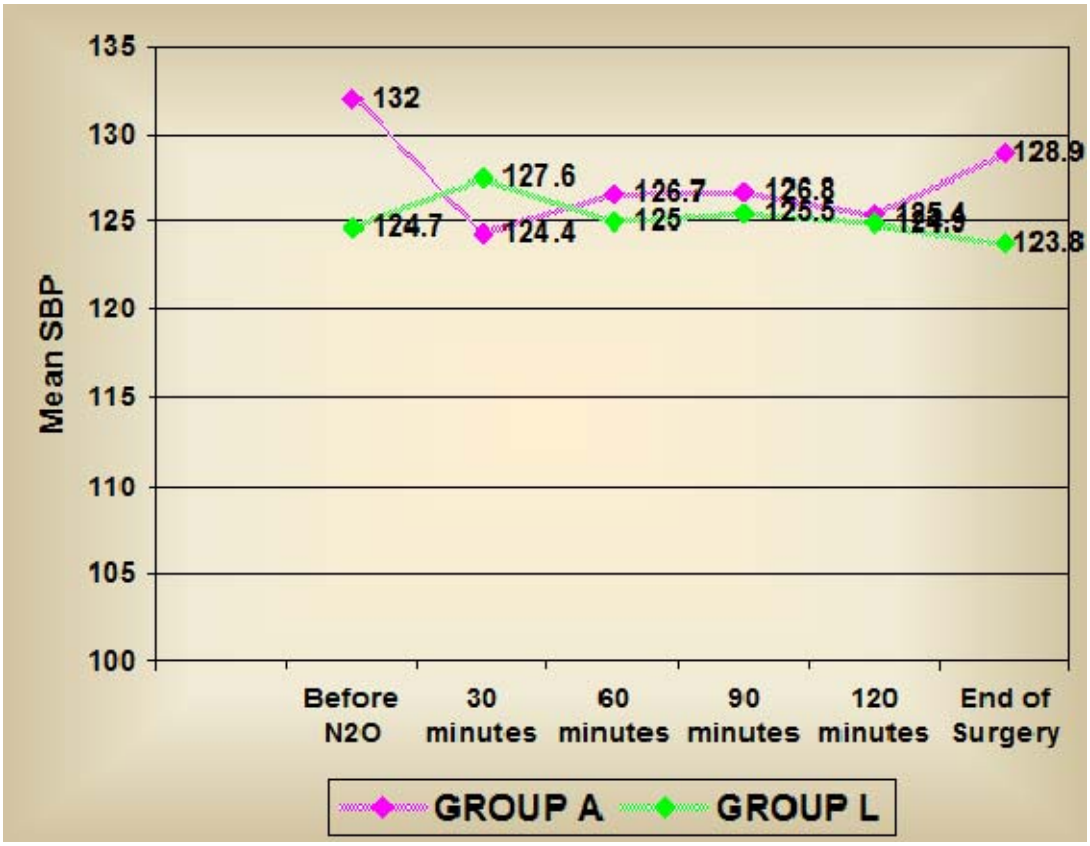

Table 5: Changes in Diastolic Blood pressure

DBP at	Diastolic B.P(mm/Hg) in				'p'	Significance
	Group A		Group L			
	Mean	SD	Mean	SD		
Before N2O	84.8	8.9	86.1	9.3	0.5121	Not significant
30 minutes	84.3	7.0	86.7	8.6	0.3999	Not significant
60 minutes	83.0	10.8	86.8	10.5	0.2319	Not significant
90 minutes	85.2	6.8	83.2	8.7	0.5984	Not significant
120 minutes	85.9	8.9	83.4	6.4	0.5392	Not significant
End of surgery	87.6	8.3	84.8	5.3	0.1058	Not significant

Diastolic Blood pressure showed no statistical significant difference between two groups.

CHANGES IN DIASTOLIC B.P.

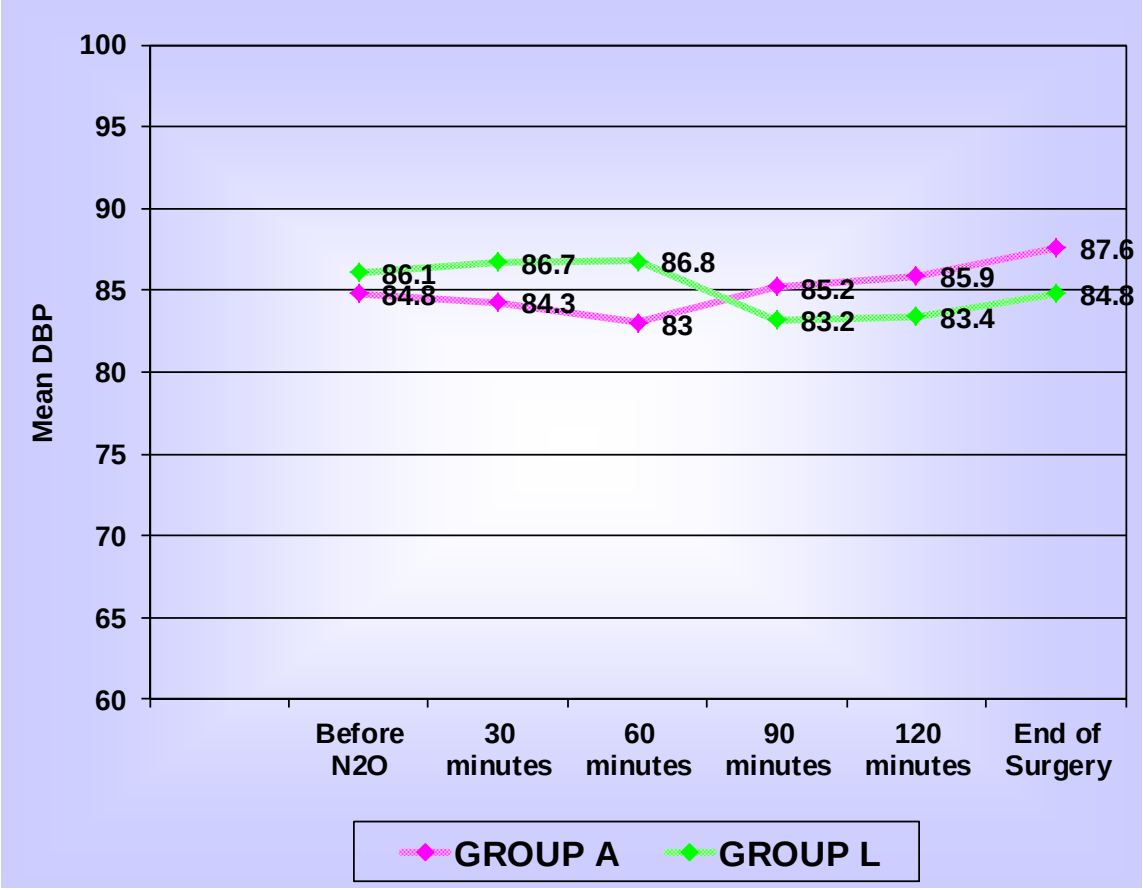

Table 6: Changes in pulse rate

Pulse rate at	Group A		Group L		'p'	Significance
	Mean	SD	Mean	SD		
Before N2O	100.2	15.1	95.2	9.0	0.2805	Not significant
30 minutes*	97.4	9.6	94.2	7.2	0.156	Not significant
60 minutes	83.0	10.8	86.8	10.5	0.2319	Not significant
90 minutes	85.2	6.8	83.2	8.7	0.5984	Not significant
120 minutes	85.9	8.9	83.4	6.4	0.5392	Not significant
End of surgery	86.4	5.1	84.4	5.6	0.3142	Not significant

Pulse rate showed no statistical significant difference between two groups.

CHANGES IN PULSE RATE

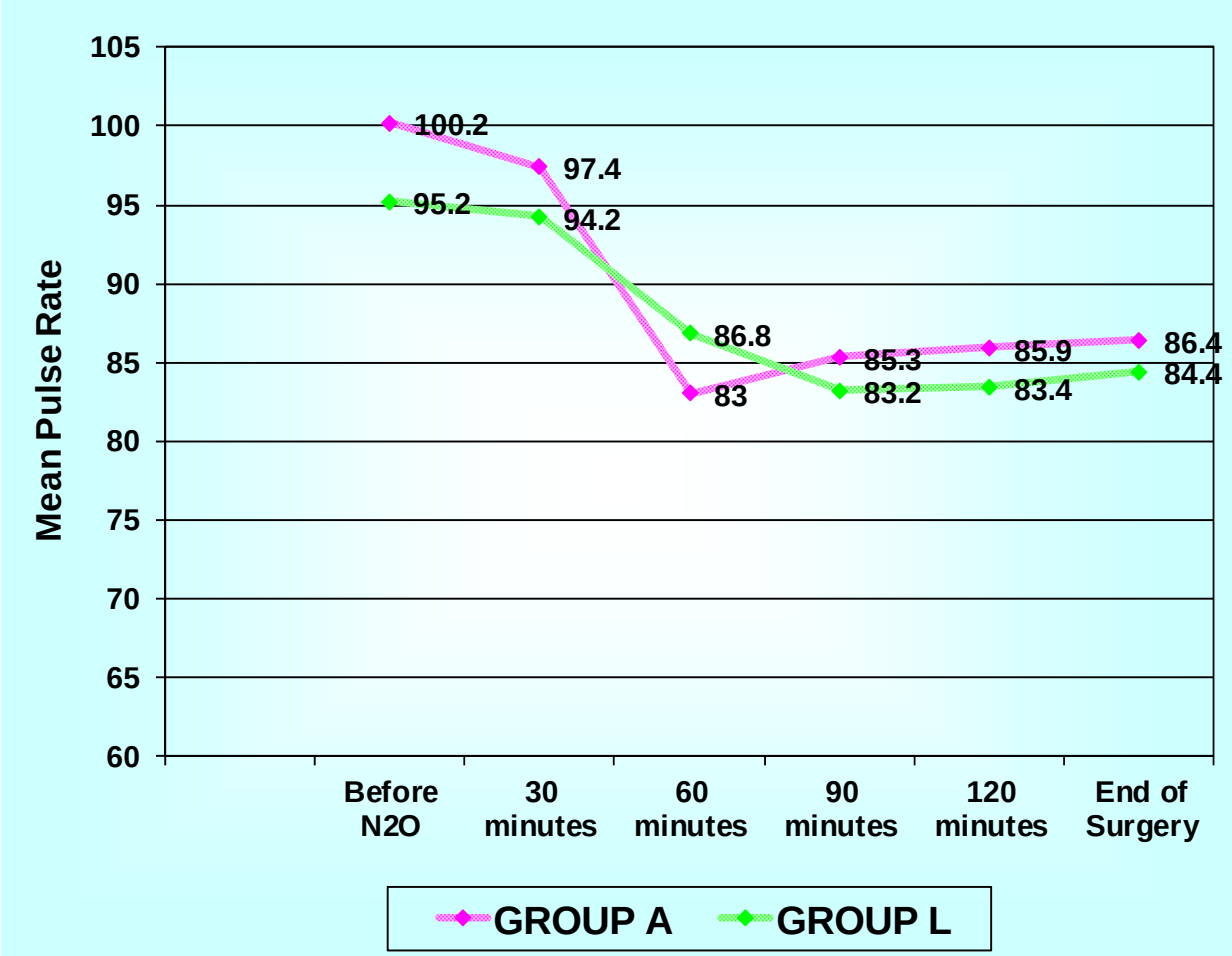

Table 7: Changes in ETCO2

ETCO2 at	ETCO2 in				'p'	Significance
	Group A		Group L			
	Mean	SD	Mean	SD		
Before N2O	31.1	3.1	32.5	2.9	0.1344	Not significant
30 minutes	31.8	3.1	32.6	3.1	0.4393	Not significant
60 minutes	32.0	3.2	31.7	3.3	0.8906	Not significant
90 minutes	31.2	2.8	32.8	2.9	0.1081	Not significant
120 minutes	31.7	3.0	31.7	3.0	0.9142	Not significant
End of surgery	32.6	3.7	31.6	2.8	0.4704	Not significant

Regarding ETCO2 p value is insignificant

CHANGES IN ETCO2

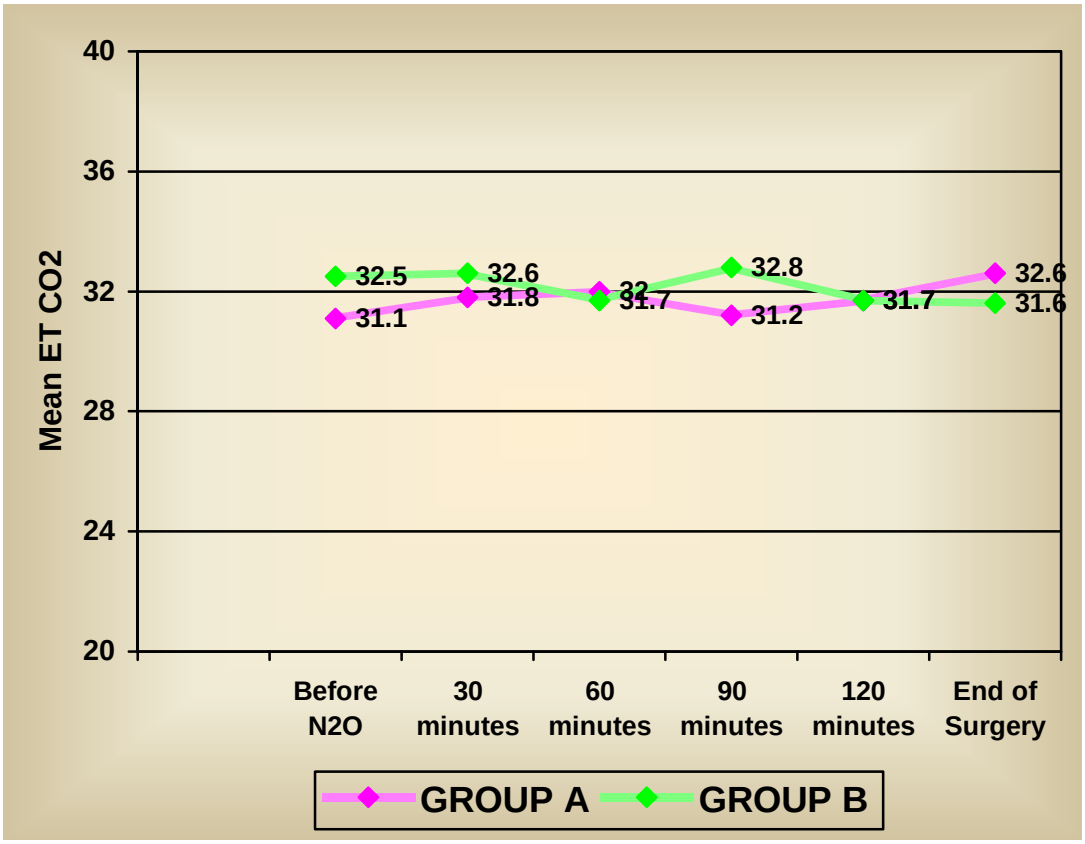

Table 8: Changes in SPO2

SPO2	SPO2 in				'p'	Significance
	Group A		Group L			
	Mean	SD	Mean	SD		
Before N2O	99.4	0.9	99.5	0.5	0.9824	Not significant
30 minutes	99.4	0.8	99.3	0.7	0.4361	Not significant
60 minutes	99.2	0.7	99.3	0.7	0.5059	Not significant
90 minutes	99.3	0.7	99.3	0.7	0.7664	Not significant
120 minutes	99.2	0.7	98.8	0.6	0.1021	Not significant
End of surgery	99.0	1.1	99.0	0.5	0.2844	Not significant

SPO2 shows no significant difference between two groups.

CHANGES IN SPO2

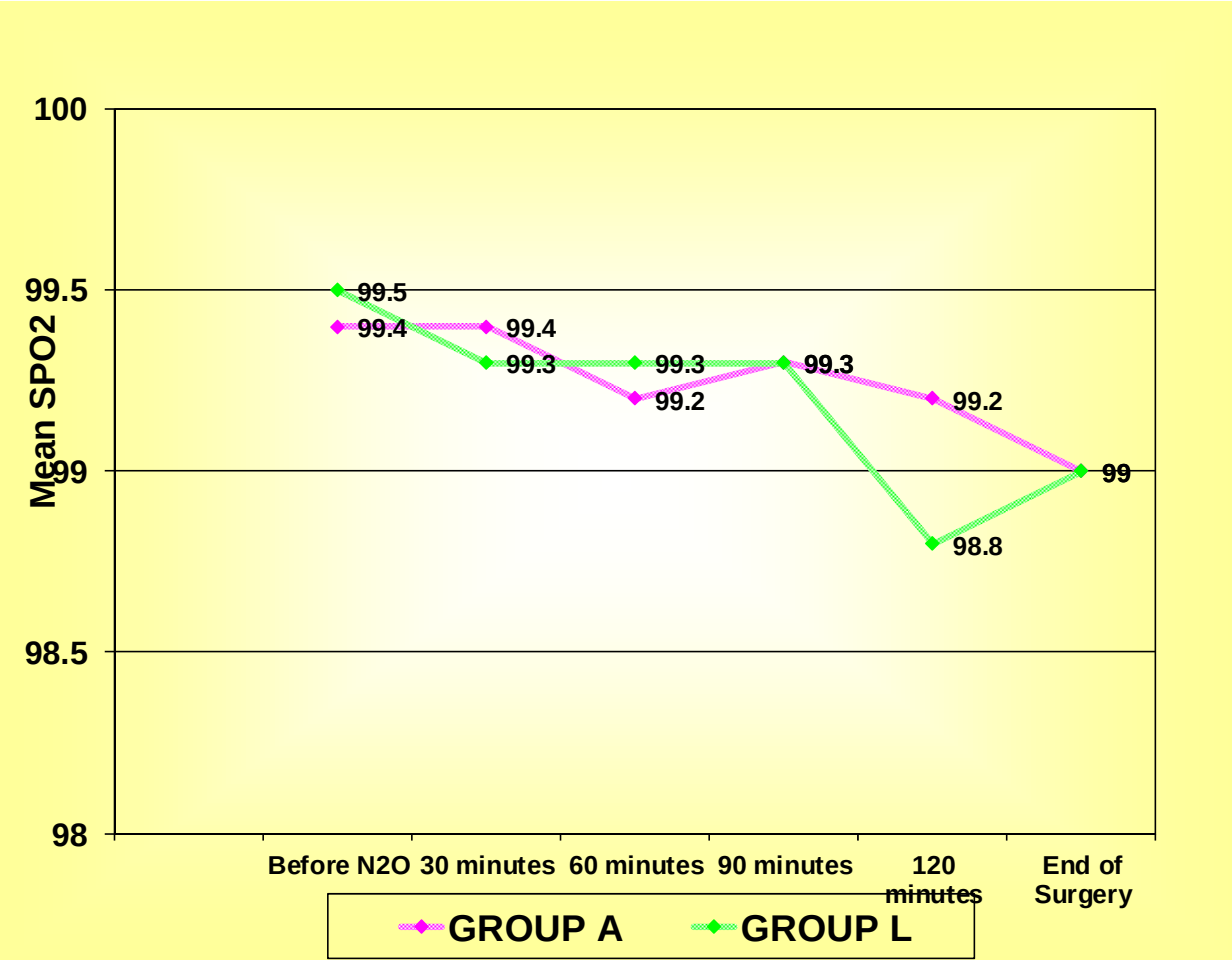

Table 9: Changes in cuff pressure

Cuff pressure at	Cuff pressure in				'p'	Significance
	Group A		Group L			
	Mean	SD	Mean	SD		
Before N2O	20	-	20	-	-	-
30 minutes	22.7	1.7	20.3	0.5	0.0001	**Significant**
60 minutes	25.4	2.3	20.7	0.8	0.0001	**Significant**
90 minutes	27.6	2.1	19.9	0.7	0.0001	**Significant**
120 minutes	29.7	2.2	19.1	0.7	0.0001	**Significant**
End of surgery	31.5	1.8	18.8	0.7	0.0001	**Significant**

P value is < 0.0005 which shows there is a statistical difference between the two Groups regarding cuff pressure.

CHANGES IN CUFF PRESSURE

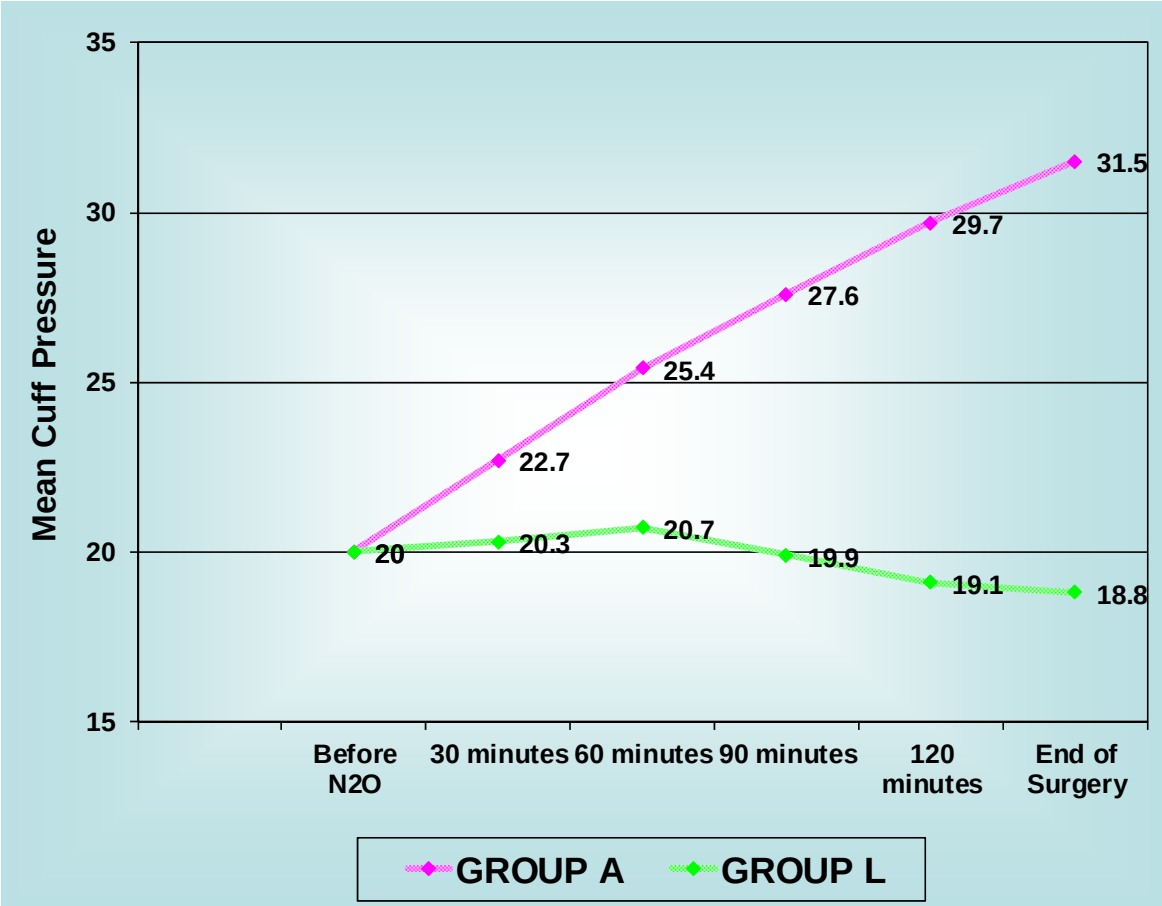

Table 10: Duration of Anaesthesia

Group	Duration of Anaesthesia (in minutes)		
	Range	**Mean**	**SD**
Group A	120-180	135.5	16.2
Group L	120-165	135.6	12.9
'p'	**0.755 (not significant)**		

Duration of anaesthesia is comparable between two groups.

DURATION OF ANAESTHESIA

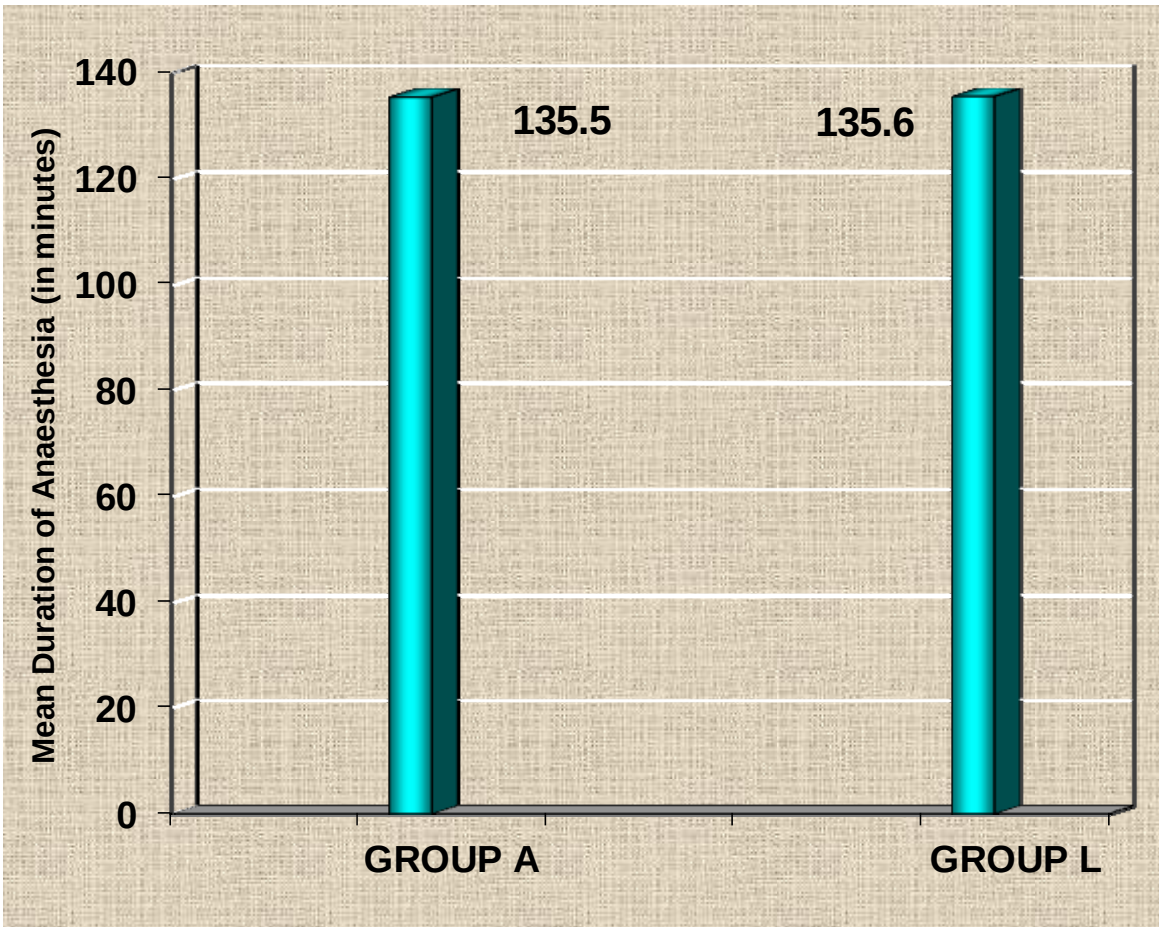

Table 11: Endotracheal tube tolerance at time of extubation

ET tube tolerance at time of extubation	Group A		Group L	
	No	%	No	%
Yes	15	60	7	28
No	10	40	18	72
'p'	**0.0461 (significant)**			

Endotracheal tube tolerance was better in group L in comparision to group A.

ENDOTRACHEAL TUBE TOLERANCE AT TIME OF EXTUBATION

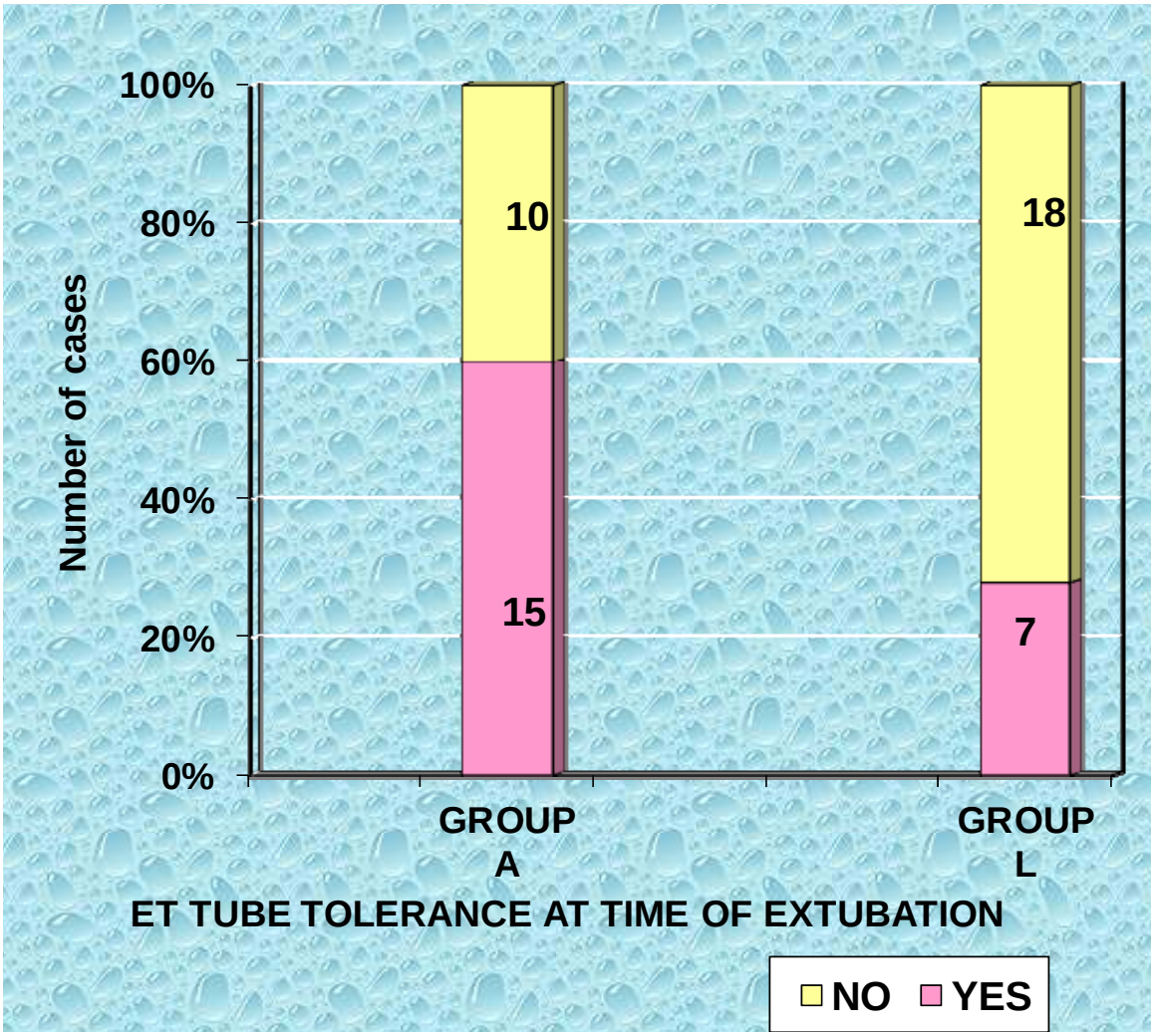

Table 12: Vital parameters at the time of extubation

At the time of extubation	Group A		Group L		'p'
	No	**%**	**No**	**%**	
Pulse rate	117.5	7.2	105.9	12.3	**0.0003 Significant**
SBP (mm/Hg)	149.0	7.5	130.6	10.5	**0.0001 Significant**
DBP (mm/Hg)	95.1	6.6	91.4	9.1	**0.1795 Not significant**

Pulse rate, systolic blood pressure shows significant difference at the time of extubation whereas diastolic blood pressure is similar in both groups.

PULSE RATE/ SBP / DBP AT EXTUBATION

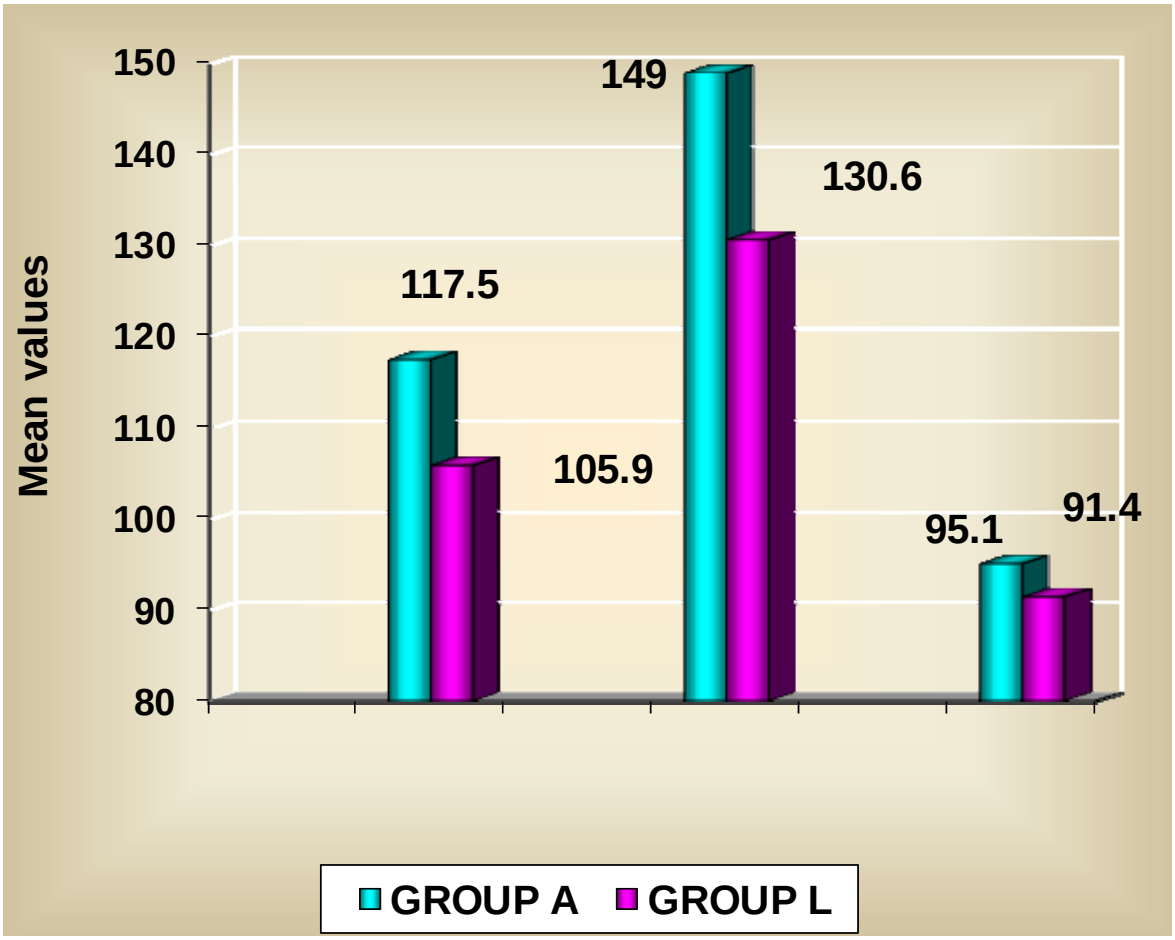

TABLE 13: CUFF VOLUME

	Group A		Group L		'p'	Significance
	Mean	**SD**	**Mean**	**SD**		
Volume inflated.(ml)	9.56	2.26	9.4	1.94	0.833	**Not significant**
Duration of anaesthesia (min.)	135.5	16.2	135.6	12.9	0.755	**Not significant**
Volume deflated (ml).	12.34	2.26	8.5	1.94	0.0001	**Significant**

Volume of the cuff contents deflated at the time of extubation shows Statistical significant difference between Group A and Group L.

CUFF VOLUME

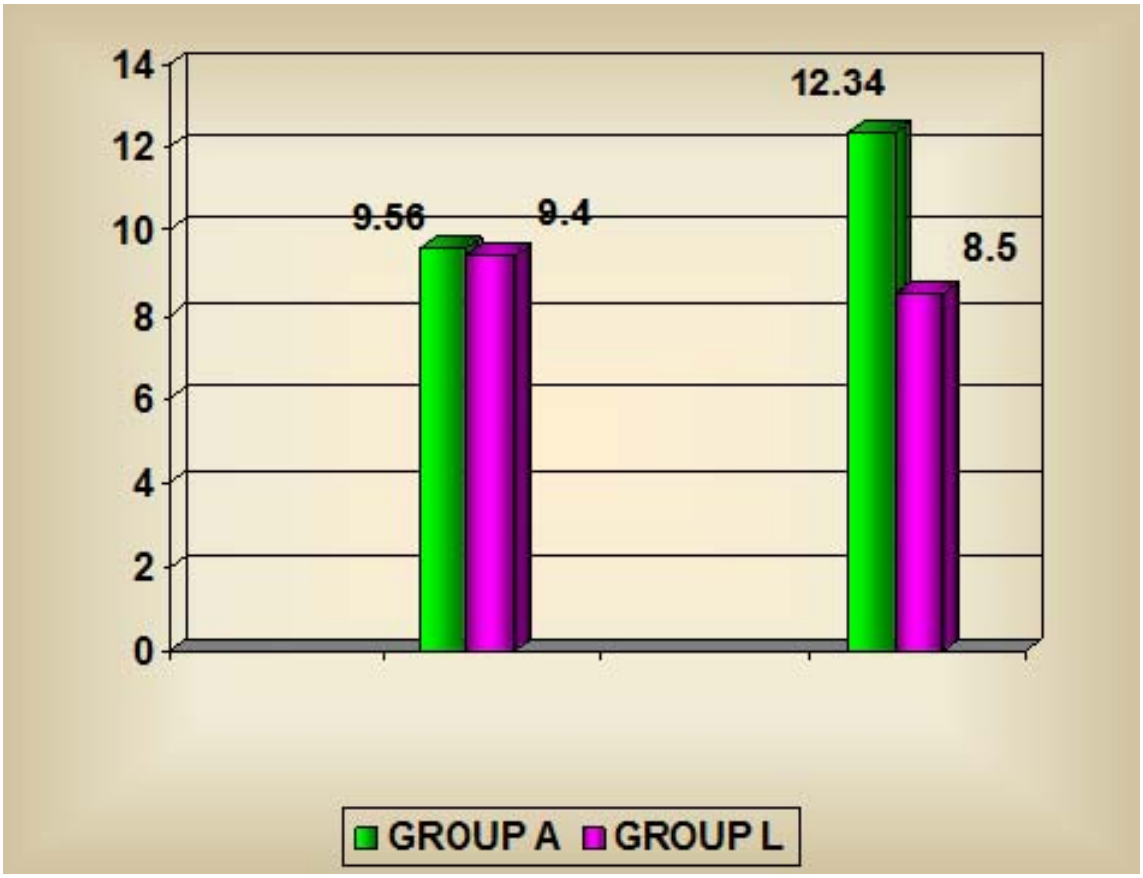

Table 14: Sore throat

Sore throat at	No. of cases in								'p'
	Group A				Group L				
	Yes		No		Yes		No		
	No	%	No	%	No	%	No	%	
30 minutes	5	20	20	80	2	8	23	92	0.2087 (Not significant)
24 hours	10	40	15	60	2	8	23	92	**0.0204 Significant**

After 24 hours of extubation, occurrence of sore throat was less in group L.

SORE THROAT

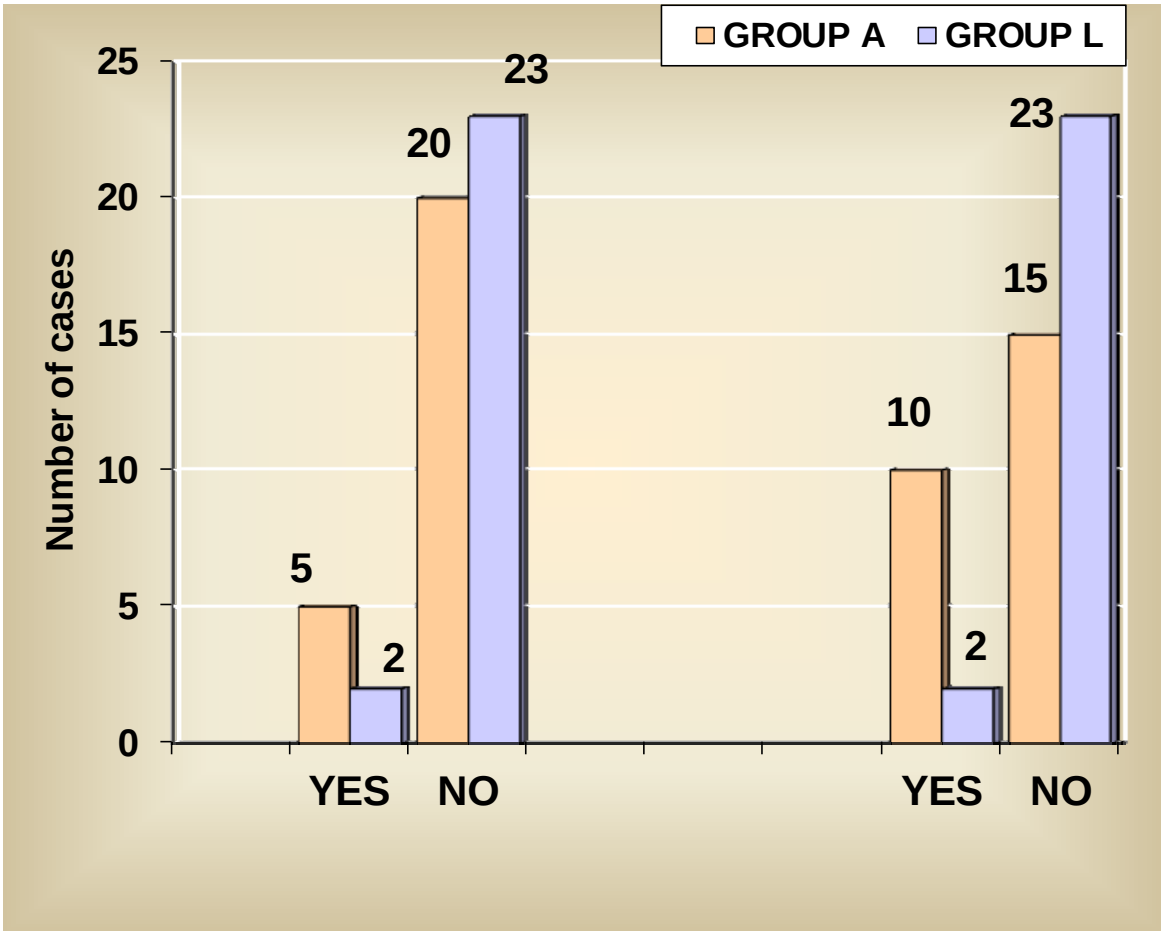
GROUP A
GROUP L
Number of cases
25
20
15
10
5
0
5
2
20
23
10
2
15
23
YES
NO
YES
NO

Table 15: Coughing

Coughing at	Group A				Group B				'p'
	Yes		No		Yes		No		
	No	%	No	%	No	%	No	%	
30 minutes	6	24	19	76	4	16	21	84	0.7237 Not significant
24 hours	-	-	25	100	-	-	25	100	-

Incidence of coughing is similar in both groups.

Table 16: Hoarseness

Hoarseness at	Hoarseness in								'p'
	Group A				Group L				
	Yes		No		Yes		No		
	No	%	No	%	No	%	No	%	
30 minutes	10	40	15	60	4	16	21	84	0.1153 Not significant
24 hours	12	48	13	52	5	20	20	80	0.0733 Not significant

Hoarseness is similar in both groups.

DISCUSSION:

Nitrous Oxide is normally used in patients undergoing general anaesthesia with controlled ventilation. During anaesthesia, nitrous oxide diffuses more rapidly into air filled cuff which depends on partial pressure across PVC cuff membrane. When cuff pressure exceeds the tracheal mucosal capillary pressure(>30mmHg),tracheal erosion occurs which causes cough &sore throat postoperatively.

Activation of rapidly acting stretch receptors in trachea will stimulate cough so that smooth extubation is not possible which will increase the systemic vascular resistance & agitation.

V.V.Ravichandran et al demonstrated that instillation of alkalinized lignocaine into the cuff reduces these complication. Use of buffered lignocaine increases the diffusion of local anesthetics & also decreased the amount of lignocaine used. Absorption of lignocaine into systemic circulation is very less. Since tracheal mucosal epithelium is very thick ,absorption rate is very low with prolonged local action.

Navvaro et al demonstrated in his study in which addition of 1ml of sodium bicarbonate (7.4%) to 19ml of lignocaine hydrochloride changes the acidic pH(6.92) to alkaline pH (7.43),similar dilution is used in my study.

Sconzo et al reported that endotracheal tube cuff filled with lignocaine diffuses across the PVC membrane & quantity of diffusion depends on the concentration of lignocaine and the duration of contact with tracheal mucosa. These can be done for the patient in ventilatory support for prolonged period as proven by previous study. Requirement of narcotics is less for the patient recieving buffered lignocaine.

COMPARISION OF RESULTS:

In this study GROUP A(ROOM AIR) & GROUP L (ALKALINIZE LIGNOCAINE) patients were comparable with age, sex, ASA risk, duration of surgery which is similar to the study done by Navvaro et al & Estebe et al. In my study there is a no significant changes intraoperative systolic blood pressure pulse rate, diastolic pressure between two groups. This was similar to the study done by Navvaro et al.

In contrast, Huang et al reported less incidence of hemodynamic changes intraoperatively compared to saline group in which there is an increase in systolic mean arterial pressure compared to other.

Pressure exerted by pilot balloon is the indirect measure of the pressure exerted by the cuff on the tracheal mucosa In regards to cuff pressure, in my study there is a significant increase in cuff pressure in Group A compared to Group L($p=0.0001$) in which there is a decrease in cuff pressure after 90

minutes of anaesthesia similar to study done by Navvaro et al. He demonstrated the significant rise in cuff pressure in air group after 30 minutes of anaesthesia compared to lignocaine group he studied.They have done an another study in which they have compared with saline instead of air. Saline group shows significant rise in cuff pressure compared to lignocaine group in which cuff pressure remains constant.

Volume of air/lignocaine removed after extubation shows significant difference in my study .Though I have injected approximately 9.5ml of air/ lignocaine after intubation, volume of air &lignocaine deflated during extubation was 12ml & 8.5ml respectively(p=0.0001).This was similar to study done by Navvaro et al who injected 5 & 6.5 ml of air & lignocaine respectively. The mean volume of air & lignocaine removed was 8 & 5 ml respectively.

Shroff et al found out that volume of air deflated is more related to volume of air inflated as compared to decrease in volume of buffered lignocaine .This increase in volume of air denotes that Nitrous oxide diffuses through the air filled cavities which doesn't occur with fluid filled cavities. Decrease in the volume of alkalinized lignocaine denotes the permeability of buffered lignocaine to polyvinyl chloride endotracheal tube wall. Diffusion of lignocaine anaesthetize the tracheal mucosa thereby reducing the airway irritation.

Hirota et al studied that tracheostomy cuff filled with lignocaine reduces airway discomfort by 50%. In my study incidence of agitation during extubation was lower in Group L compared to Group A(p=0.0461).

This result was similar to the study done by V.V.Ravichandran et el who demonstrated smooth extubation in patients with hyper-reactive airway receiving intra cuff buffered lignocaine compared to air& normal saline.

Similarly Navvaro et al also demonstrated that incidence of agitation during extubation was lower in buffered lignocaine group compared to air group.

Behzadi et al reported the efficiency of intra cuff lignocaine by promoting smooth emergence in children undergoing adenotonsillectomy compared to saline group. In my study there is a maintanence of systolic blood pressure & pulse rate in Group L compared to Group A in which there is a significant increase in above parameters at the time of extubation.

But the study done by Navvaro et al shows rise in systolic blood pressure at time of extubation both in air & lignocaine group(air >lignocaine) .The diastolic pressure and the heart rate doesnot Change.

Estebe et al found out that there was no significant difference in heart rate & blood pressure at time of extubation in both air & lignocaine group.

Behzadi et al reported the efficiency of intra cuff lignocaine by promoting smooth emergence in children undergoing adenotonsillectomy compared to saline group. In my study there is a maintanence of systolic blood pressure & pulse rate in Group L compared to Group A in which there is a significant increase in above parameters at the time of extubation.

But the study done by Navvaro et al shows rise in systolic blood pressure at time of extubation both in air &lignocaine group(air >lignocaine) .The diastolic pressure and the heart rate doesnot Change.

Estebe et al found out that there was no significant difference in heart rate & blood pressure at time of extubation in both air & lignocaine group.Parameters at the time of extubation when intra cuff saline or lignocaine is injected.

Venkatesan et al showed that there was no significant difference in haemodynamic parameters between I.V. lignocaine and intra cuff 4% lignocaine at the extubation when he conducted study in neurosurgical patients. Altintas et al found out that endotracheal tube cuff filled with 10% lignocaine produces less hemodynamic responds during extubation compared to intra cuff saline groups.

Huang et al concluded that hemodynamic changes in systolic pressure was higher in 4% lignocaine group compared to buffered lignocaine (4% lignocaine + 1ml of sodium bicarbonate). This result was similar to my result. Regarding sore throat in my study there was no significant difference between air (group A) and lignocaine group(L) after 30 minutes of extubation. But after 24hours of extubation incidence of sore throat was significantly lower in group L compared to group A.

This was similar to study done by Navvaro et al who demonstrated that incidence of sore throat was lower in incidence after 24 hours of extubation when he compared intra cuff air and lignocaine.

There was no notable difference between two groups immediately after extubation. V.V.Jaichandran et al concluded in his study that the incidence of sore throat was lower to in lignocaine group compared to air group.

Lecorre et al recorded that incidence of sore throat was lower in buffered lignocaine group compared to air group during the postoperative period.

Soltani et al concluded in his study, by comparing intra cuff alkanized lignocaine and intra venous lignocaine at the end of surgery decreases the incidence of sore throat in both groups.

Huang et al observed from his study that there is a 35% of decrease in incidence of sore throat in buffered lignocaine group compared to normal saline group.

Above study results were similar to my results but according to Porter et al who observed that there was no significant difference in sore throat by comparing intra cuff lignocaine, saline and air group.

Rapidly adapting stretch receptors present over the tracheal mucosa are stimulated by endotracheal tube intubation and hyperinflation of the cuff during general anaesthesia using nitrous oxide which was responsible for the cough after extubation. Anesthetizing the tracheal mucosa with lignocaine prevents this complication during immediate and late postoperative period.

The incidence of coughing in postoperative period does not produce any significant difference in my study this was similar to the study done by Navvaro et al.

Soltani et al demonstrated that intra cuff lignocaine decreases the incidence of postoperative cough compared to other groups (different application of lignocaine).

Sagheer et al compared intra cuff 4% lignocaine and air group and found out that there was decrease in the frequency of coughing at extubation and postoperative period lignocaine group compared to other.

According to Fagan et al incidence of coughing was lower in lignocaine group in the initial post extubation period when compared to saline or lignocaine group.

Above studies showed that incidence of cough was lower in lignocaine group but in my study there was no significant reduction in cough between two groups I compared.

Incidence of hoarseness was low but does not produce any significant difference between air and lignocaine group in my study. This was similar to study done by navvaro et al.

Le corre et al reported less incidence of hoarseness in intra cuff buffered lignocaine group compared to intra cuff air group.

Shroff et al observed in his study that there was decrease in incidence of hoarseness in intra cuff lignocaine group compared to intra cuff air group.

Shin et al concluded in his study that the use of intra cuff alkanized lignocaine decrease the incidence of hoarseness compared to intra cuff air group.

SUMMARY OF THE STUDY:

This study was carried out at Govt. Rajaji hospital, Madurai in 50 patients of age 18-65 years of ASA I,II,&III who underwent abdominal &breast surgeries under general anaesthesia with N2O.They were divided into two groups 25 patients in each group.

GROUP A:Inflated with air upto pressure of 20 cm of water.

GROUP L: Inflated with alkalinized lignocaine(2% lignocaine : sodium bicarbonate 19:1 up to pressure of 20 cm of water .

All Patients were pre medicated with Inj. Glycopyrolate 0.2 mg intramuscularly 45 minutes before surgery. Monitors were connected. Intravenous cannula secured and connected to i. v fluids. Patient were pre oxygenated with 100% O2 for 3 minutes. Patient is induced with Inj. fentanyl 2 micrograms/kg ,Inj. propofol 2mg/Kg i.v, Inj. Atracurium 0.5 mg/kg and intubated with 7.5 mm(females), 8mm(males) endotracheal tube. Endotracheal tube was inflated according to study group.

Bilateral air entry checked and connected to closed circuit. Patient is maintained with N2O : O2 2:2, Inj. fentanyl 1 micrograms/kg, inj. atracurium in titrated doses Baseline cuff pressure before N2O administration is monitored. Cuff pressure is recorded every 30 minutes thereafter .At the end of surgery, cuff pressure, tube tolerance, volume of air/alkalinized lignocaine deflated

during extubation were noted. After adequate attempts of respirations, patient is reversed with inj.Glycopyrolate 10 mics/kg , inj. neostigmine 40 mics/kg and extubated after deflation of the cuff.

INTRAOPERATIVELY following parameters were monitored.

1. PR, BP, SPO, ETCO2, Cuff pressure were noted before nitrous oxide administration ,&every 30 minutes till the end of surgery.
2. Tube tolerance ,BP, PR, volume of air /lignocaine deflated were noted.

POSTOPERATIVELY following parameters were noted: Coughing, sore throat, hoarseness were monitored 30 minutes after surgery in post anaesthesia care unit & 24 hours later. Pain was assessed using visual analogue scale.

This study showed that

1. Intracuff injection of alkalinized lignocaine reduces the cuff pressure during general anaesthesia with N2O compared to cuff inflated with air in which there is a gradual increase in cuff pressure.
2. Incidence of rise in systolic blood pressure during extubation was more in GROUP A compared to GROUP L.
3. ET tube tolerance during extubation was more with GROUP L compared to

GROUP A.

4. Volume of lignocaine deflated during extubation was less than the infleated volume. But volume of air deflated was more than the inflated volume.
5. Incidence of sore throat was similar in immediate & less in late postoperative period in intra cuff lignocaine group (GROUP L) compared to

CONCLUSION:

From the data &statistical analysis, intracuff injection of alkalinized lignocaine instead of room air reduces the cuff pressure which provide better endotracheal tube tolerance with less hemodynamic changes during extubation & less incidence of sorethroat in post operative period.

BIBLIOGRAPHY:

1. Miller's anesthesiology seventh edition, Morgan's clinical anesthesiology fourth edition
2. Ellis'anatomy eighth edition.
3. Text book of pharmacology-K.D .Tripathi sixth edition.
4. Goodman & Gillman text book of pharmacology.
5. Dorsch & Dorsch's understanding anaesthesia equipment fifth edition.
6. Navarro RM, Baughman VL. Lidocaine in the endotracheal tube cuff reduces postoperative sore throat. J Clin Anesth.1997;9(5):394-7.
7. Huang CJ, Hsu YW, Chen CC, et al. Prevention of coughing induced by endotracheal tube during emergence from general anaesthesia--a comparison between three different regimens of lidocaine fi lled in the endotracheal tube cuff. Acta Anaesthesia scandinacica. 1998;36(2):81-6.
8. Fagan C, Frizelle HP, Laffey J, Hannon V, Carey M. The effects of intracuff lidocaine on endotracheal-tube-induced emergence phenomenon after general anaesthesia. Anesth Analg.2000;91(1):201-5.
9. Altintas F, Bozkurt P, Kaya G, Akkan G. Lidocaine 10% in the endotracheal tube cuff: blood concentrations, haemodynamic and clinical effects. Eur J Anaesthesiology. 2000;17(7):436-42.
10. Dollo G, Estebe JP, Le Corre P, Chevanne F, Ecoffey C, Le Verge R. Endotracheal tube cuffs fi lled with lidocaine as a drug delivery system:

invitro and in vivo investigations. Eur J Pharm Sci. 2001;13(3):319-23.

11. Estebe JP, Dollo G, Le Corre P, et al. Alkalinization of intracuff lignocaine improves endotracheal tube-induced emergence phenomenon. Anesth Analg. 2002;94(1):227-30.
12. Soltani HA, Aghadavoudi O. The effect of different lignocaine application methods on postoperative cough and sore throat. J Clin Anesth. 2002;14(1):15-8.
13. V.V.Ravichandran,V.Jegadeesh,et al.Intracuff buffered lignocaine versus saline or air SAJAA 2009 15(2) :11-14.
14. Le Corre P,Dollo G, Chevanne F, Ecoffey C.Alkalinization of intracuff lignocaine : efficacy & safety. Journal of anaesthesia analgesia 2005.
15. Shroff PP, Patil V.Efficacy of cuff inflation media to prevent post intubation-related emergence phenomenon:air,saline& alkalinised lignocaine.
16. Shin JC, Kim KS, Kim YJ,Choi WJ, Koo MS.The effects of the alkalinization of intracuff lignocaine after Nitrous Oxide Anaesthesia. Korean journal of anesthesiology 2008.
17. Venkatesan T,Korula G.A comparative study between the effects of 4% endotracheal tube cuff lignocaine & 1.5mg/Kg I.V.lignocaine on coughing & haemodynamics during extubation in neurosurgical patients. Journal of neurosurgery anesthesiology 2006./

STUDY PROFORMA:

COMPARATIVE STUDY OF THE CUFF PRESSURE BETWEEN AIR AND ALKALINISED LIGNOCAINE IN GENERAL ANAESTHESIA.

NAME: AGE: SEX: I.P.NO:

ASA: MPG: DIAGNOSIS:

PROCEDURE: DURATION OF SURG:

LAB.TEST:

Hb%,

BLOOD SUGAR ,UREA ,CREATININE .,

S.ELECTROLYTES,

ECG ,CHEST X-RAY (P.A. VIEW)

STUDY GROUP:

GROUP A- ROOM AIR

GROUP L-ALKALINISED LINOCAINE

INTRA OP:

(TIME IN MINS)

	BEFN20 ADMIN	30	60	90	120	END OF SURG
PR						
BP						
SPO2						
PCO2						
CUFF PR						

DURATION OF ANAESTHESIA:

TIME OF EXTUBATION:

1.presence of agitation dur.spont ven(yes/no)-

2.PR- BP-

3.vol.of air/lignocaine on deflation:

PACU:

	30 MINS	24 HRS (VAS)
COUGHING		
SORE THROAT		
HOARSENESS		

GROUP A

Sl. No.	GROUP	NAME	AGE (years)	SEX	I.P.NO	ASA	MPG	DURATION OF ANAESTHESIA (MINS)	volume of A/L inflated(ml)	BEFORE N2O						30 mins						60 mins					
										systolic Blood pressure (mmHg)	diastolic Blood Pressure(mmHg)	Pulse rate(min)	ETCO2	SPO2(%)	CUFF Pressure(cm H2O)	systolic blood Pressure(mmHg)	diastolic Blood Pressure (mmHg)	Pulse Rate(min)	ETCO2	SPO2(%)	CUFF Pressure (cm H2O)	systolic Blood Pressure(mmHg)	diastolic Blood Pressure(mmHg)	Pulse Rate(min)	ETCO2	SPO2(%)	CUFF Pressure(cmH2O)
1	air	Mariammal	40	F	89840	III	I	150	8	120	90	92	32	99	20	110	90	88	33	99	22	115	76	95	28	99	23
2	air	Rajeshwari	22	F	91819	I	I	130	7	115	80	112	30	98	20	108	86	90	35	100	23	120	86	85	26	99	25
3	air	Senthil	25	M	91147	I	I	140	11	125	90	125	34	100	20	115	80	89	32	98	21	120	80	94	30	99	22
4	air	Ayyaperumal	42	M	34215	II	I	120	12	134	90	122	28	98	20	126	76	94	30	99	20	130	72	102	33	100	24
5	air	Kannan	46	M	92853	I	I	124	12	142	92	119	33	97	20	132	78	95	28	100	22	128	80	98	35	100	25
6	air	Vimala devi	20	F	4490	II	I	129	7	128	85	95	35	99	20	112	94	72	30	100	21	105	65	80	38	100	23
7	air	Kalyani	36	F	765	II	I	135	7	133	82	88	32	100	20	119	85	98	33	100	23	128	85	84	32	100	26
8	air	Indrani	40	F	708	II	I	122	8	118	65	75	37	100	20	135	90	90	35	100	22	122	90	80	30	100	29
9	air	Nachiar	48	F	89176	I	I	142	8	152	108	116	30	100	20	142	93	94	30	99	23	150	100	113	33	100	25
10	air	Dinesh	22	M	91712	I	I	125	7	130	86	84	28	100	20	122	76	90	32	98	21	145	94	108	33	100	23
11	air	Ravikumar	29	M	2072	I	I	122	12	120	80	78	25	99	20	132	92	112	25	99	22	129	95	94	30	100	27
12	air	Shakil begum	43	F	1223	II	I	132	8	130	75	88	27	100	20	126	78	96	36	99	21	130	87	99	28	98	23
13	air	Karuppaiah	60	M	90101	III	I	180	13	140	95	92	29	100	20	132	90	95	38	99	24	122	90	92	30	98	28
14	air	Karthikeyan	25	M	2563	I	I	120	12	129	85	89	33	100	20	122	86	92	33	99	26	118	89	88	33	98	29
15	air	Mallika	50	F	4791	II	I	130	8	132	75	94	35	100	20	110	70	94	30	99	21	99	65	70	30	98	22
16	air	Pandian	48	M	89536	III	I	140	12	125	82	100	29	99	20	132	92	110	35	100	23	148	95	108	29	99	25
17	air	Sakthivel	36	M	2560	I	I	180	12	122	85	92	32	99	20	144	90	120	34	100	24	123	78	102	33	99	27
18	air	Vijayalatha	54	F	3761	II	I	138	8	140	78	108	30	98	20	122	80	98	30	100	22	120	75	89	34	99	23
19	air	Saroja	58	F	6424	II	I	150	8	132	79	98	26	99	20	117	78	104	28	100	21	125	76	90	32	99	24
20	air	Mariappan	46	M	6440	I	I	120	12	122	70	80	33	100	20	110	90	96	33	100	25	124	79	95	30	99	29
21	air	Amutha	21	F	9227	I	I	125	7	145	90	114	32	100	20	109	85	102	34	97	24	125	76	75	33	99	26
22	air	Janaki	52	F	5471	II	I	138	8	134	85	98	35	100	20	138	76	108	28	100	26	142	108	100	38	99	28
23	air	Poovalagi	49	F	4298	II	I	135	8	155	89	120	34	100	20	142	93	104	33	100	22	132	86	95	32	99	24
24	air	Arumugam	25	M	5429	I	I	120	12	145	87	122	29	100	20	132	85	98	32	100	25	144	83	92	39	99	28
25	air	Arumugam	55	M	8336	I	I	140	12	132	96	103	29	100	20	120	75	106	28	100	23	124	66	88	30	99	26

90 mins						120 mins						END OF SURGERY						TIME OF EXTUBATION					COUGH		S T		HOARSE	
systolic Blood Pressure(mmHg)	diastolic Blood Pressure(mmHg)	Pulse Rate(min)	ETCO2	SPO2(%)	CUFF Pressure (cmH2O)	systolic Blood Pressure(mmHg)	diastolic blood pressure(mmHg)	Pulse rate(min)	ETCO2	SPO2(%)	CUFF Pressure (cmH2O))	systolic Blood Pressure(mmHg)	diastolic Blood Pressure(mmHg)	PULSE RATE (min)	ETCO2	SPO2(%)	CUFF Pressure (cmH20)	AGITATION	Pulse rate (min)	Systolic blood pressure (mmHg)	Diastolic blood pressure(mmHg)	volume of A/L deflated (ml)	30 mins	24hrs	30mins	24hrs	30mins	24hrs
130	85	85	35	99	25	115	83	90	33	99	27	130	74	90	29	99	30	YES	115	150	100	11	NO	NO	NO	YES	YES	YES
114	84	89	35	99	29	130	82	95	34	99	31	150	108	85	33	100	33	NO	120	140	100	10	YES	NO	NO	NO	NO	NO
123	80	84	33	99	25	120	79	89	30	99	28	122	80	90	30	99	30	YES	108	155	90	13	NO	NO	NO	YES	NO	YES
114	90	100	28	99	28	123	95	94	28	98	30	129	89	84	35	98	32	YES	122	154	89	15	NO	NO	YES	NO	NO	YES
125	85	98	29	100	27	130	85	94	25	98	29	122	85	96	32	96	32	YES	125	160	100	14	NO	NO	NO	NO	NO	NO
100	90	85	26	100	26	110	75	88	29	100	28	125	85	90	30	98	30	YES	114	142	92	10	YES	NO	NO	YES	YES	YES
138	98	99	33	100	28	130	100	94	32	99	30	120	92	99	28	99	33	NO	110	150	100	9.5	NO	NO	NO	NO	NO	YES
132	85	96	29	100	31	129	92	99	33	99	33	130	90	82	39	98	33	YES	128	155	95	10	YES	NO	NO	YES	YES	YES
133	88	90	33	100	27	132	99	95	35	100	29	126	85	94	35	99	32	YES	130	160	100	11	YES	NO	YES	YES	YES	YES
144	85	100	35	100	25	128	95	96	33	100	27	140	90	95	38	99	29	NO	112	140	90	10	NO	NO	NO	NO	NO	NO
143	102	108	30	98	29	132	100	102	28	100	32	132	98	86	39	100	33	YES	122	135	90	15	NO	NO	NO	NO	YES	YES
126	94	103	32	98	25	140	95	96	30	100	27	130	90	92	33	100	29	NO	110	144	86	10	NO	NO	NO	NO	NO	NO
105	75	87	29	98	30	125	85	92	32	100	32	142	90	84	32	100	33	YES	108	154	96	16	NO	NO	YES	NO	NO	YES
120	84	78	28	98	31	110	85	85	29	100	33	112	75	89	30	100	33	NO	110	145	85	15	NO	NO	NO	YES	YES	NO
125	85	85	26	100	25	144	76	95	33	100	27	128	90	86	29	100	29	YES	128	158	95	11	YES	NO	NO	YES	NO	NO
144	79	99	33	100	27	134	85	90	35	100	30	132	78	95	26	99	32	NO	122	143	93	15	NO	NO	NO	NO	NO	YES
110	86	93	34	100	29	120	76	88	37	100	32	110	90	93	29	99	33	YES	118	145	98	14	NO	NO	NO	NO	NO	NO
133	76	95	30	100	26	109	79	100	29	100	29	150	98	88	36	98	31	YES	122	158	110	11	NO	NO	YES	NO	YES	NO
118	90	87	31	100	25	142	102	95	30	100	25	123	74	82	38	97	27	NO	112	142	85	11.5	NO	NO	NO	NO	NO	NO
123	73	90	28	99	30	125	76	79	33	100	32	122	76	89	36	98	33	NO	110	139	90	15	NO	NO	NO	YES	YES	NO
140	79	112	33	99	28	110	88	103	35	99	30	140	90	80	33	100	32	YES	128	160	100	10	YES	NO	YES	NO	NO	NO
132	82	93	29	99	30	133	76	100	37	99	32	118	98	84	30	100	33	NO	114	145	90	11	NO	NO	NO	YES	YES	NO
140	90	100	32	99	26	120	80	86	28	99	28	122	85	82	29	100	30	NO	111	149	108	10.5	NO	NO	NO	NO	NO	YES
130	85	89	35	99	30	115	75	80	32	99	32	129	89	89	31	100	33	YES	125	155	100	15	NO	NO	NO	YES	YES	NO
127	80	93	33	99	27	130	85	99	33	99	30	138	90	85	34	100	33	YES	114	148	95	15	NO	NO	NO	NO	NO	YES

GROUP L																											
									BEFORE N2O						30 mins							60 mins					
Sl. No.	GROUP	NAME	AGE (years)	SEX	I.P.NO	ASA	MPG	DURATION OF ANAESTHESIA (MINS)	volume of air inflated(ml)	systolic Blood pressure (mmHg)	diastolic Blood Pressure(mmHg)	Pulse rate(min)	ETCO2	SPO2(%)	CUFF Pressure(cm H2O)	systolic blood Pressure(mmHg)	diastolic Blood Pressure (mmHg)	Pulse Rate(min)	ETCO2	SPO2(%)	CUFF Pressure (cm H2O)	systolic Blood Pressure(mmHg)	diastolic Blood Pressure(mmHg)	Pulse Rate(min)	ETCO2	SPO2(%)	CUFF Pressure(cmH2O)
1	ligno	Baskaran	23	M	4045	I	I	130	11	120	90	112	32	99	20	110	98	95	33	99	20	109	85	95	33	99	20
2	ligno	Sundar	32	M	9953	I	I	120	12	140	100	95	35	99	20	124	78	92	35	99	21	121	89	92	36	99	21
3	ligno	Kannan	42	M	9224		I	135	12	126	80	84	37	99	20	114	78	86	33	99	20	119	85	86	32	99	21
4	ligno	Kamatchi	25	M	63444	II	I	128	12	110	79	95	30	99	20	143	89	90	31	98	20	133	76	90	30	99	20
5	ligno	Ayyammal	50	F	69648	I	I	140	8	130	90	100	32	99	20	125	90	88	29	98	20	132	92	88	29	98	21
6	ligno	Ganesan	43	M	73490	II	I	160	13	122	95	88	29	99	20	143	90	111	28	98	21	140	90	111	33	98	22
7	ligno	Arockiammal	52	F	73447	II	I	150	8	135	90	92	27	100	20	133	90	99	30	98	20	129	100	99	31	98	21
8	ligno	Mariam	30	F	80948	I	I	120	8	140	100	111	31	100	20	132	77	97	31	100	20	130	100	97	33	98	20
9	ligno	Mayathevar	50	M	83449	II	I	133	12	128	100	108	35	100	20	142	89	100	35	100	20	136	76	100	36	99	21
10	ligno	Neela	60	F	2180	II	I	160	8	125	85	102	38	100	20	120	80	86	32	100	21	125	75	86	33	100	22
11	ligno	Lakshmi	51	F	83730	II	I	130	8	100	70	86	39	100	20	122	98	98	30	100	20	106	86	98	29	100	21
12	ligno	Indira	42	F	3900	II	I	145	8	115	85	92	33	100	20	142	96	103	33	100	20	132	84	103	25	100	21
13	ligno	Prema	41	F	4121	II	I	140	8	120	90	85	32	100	20	125	78	89	39	100	20	116	83	89	30	100	20
14	ligno	Nagavalli	34	F	4130	II	I	135	8	112	78	90	33	100	20	127	90	95	35	100	21	120	80	95	28	100	22
15	ligno	Manjula	51	F	6404	II	I	165	8	132	87	100	33	100	20	142	100	109	30	100	20	129	105	109	32	100	21
16	ligno	Sundaram	31	M	9208	II	I	120	11	105	70	90	35	100	20	110	80	88	31	100	20	103	63	88	33	100	20
17	ligno	Rajathi	40	F	10041	II	I	135	8	128	100	96	30	100	20	130	90	99	36	100	21	137	95	99	38	100	22
18	ligno	Pitari	23	F	12906	II	I	138	8	125	90	79	35	100	20	138	100	89	35	100	20	132	110	89	26	100	20
19	ligno	Amsalakshmi	51	F	14660	II	I	129	8	140	90	100	32	100	20	123	74	94	33	99	20	130	80	94	28	100	20
20	ligno	Manimala	52	F	14584	I	I	120	8	120	80	84	33	99	20	145	90	96	39	99	20	109	90	96	29	100	20
21	ligno	Innasi	41	M	17195	II	I	142	12	133	76	92	29	99	20	111	78	84	29	99	21	120	90	84	33	99	20
22	ligno	Nazeema	45	F	23133	II	I	125	8	140	90	94	30	99	20	129	95	92	33	99	20	127	83	92	34	99	20
23	ligno	Muniammal	24	F	23099	II	I	140	8	133	73	102	30	99	20	126	90	91	36	99	20	136	96	91	37	99	20
24	ligno	Shanthi	40	M	23103	II	I	120	12	140	90	111	33	99	20	129	75	100	32	99	21	132	79	100	33	99	22
25	ligno	Suseela	34	F	22221	I	I	130	8	98	75	92	30	99	20	105	75	84	28	99	20	121	79	84	31	99	20

90 mins						120 mins						END OF SURGERY						TIME OF EXTUBATION					COUGH		S T		HOARSE	
systolic Blood Pressure(mmHg)	diastolic Blood Pressure(mmHg)	Pulse Rate(min)	ETCO2	SPO2(%)	CUFF Pressure (cmH2O)	systolic Blood Pressure(mmHg)	diastolic blood pressure(mmHg)	Pulse rate(min)	ETCO2	SPO2(%)	CUFF Pressure (cmH2O))	systolic Blood Pressure(mmHg)	diastolic Blood Pressure(mmHg)	PULSE RATE (min)	ETCO2	SPO2(%)	CUFF Pressure (cmH20)	AGITATION	Pulse rate (min)	Systolic blood pressure (mmHg)	Diastolic blood pressure(mmHg)	volume of air deflated (ml)	30 mins	24hrs	30mins	24hrs	30mins	24hrs
110	70	84	35	99	19	106	75	85	29	100	19	110	90	84	33	100		NO	108	129	100	10	NO	NO	NO	NO	NO	NO
120	100	88	32	99	20	122	89	88	35	100	20	128	80	80	32	100	19	NO	98	132	85	11.5	NO	NO	NO	NO	NO	NO
110	83	87	33	99	19	100	80	80	36	99	18	125	90	82	36	99	18	NO	89	120	80	11	NO	NO	NO	NO	NO	NO
125	89	93	33	98	20	129	90	99	32	99	19	120	84	84	30	99	19	NO	110	136	95	11.2	NO	NO	NO	NO	NO	NO
122	85	90	38	98	20	123	85	85	38	99	19	120	80	90	31	99	18	YES	122	140	100	7	YES	NO	NO	NO	YES	YES
130	90	95	31	100	21	122	88	90	30	99	20	110	79	89	29	99	19	NO	109	129	98	12	NO	NO	NO	NO	NO	NO
123	67	88	30	100	20	130	89	100	33	99	20	135	86	86	27	99	20	YES	125	145	110	7	NO	NO	NO	NO	NO	YES
134	88	90	30	100	20	140	90	102	32	99	19	115	75	84	35	98	19	NO	100	128	87	7.5	NO	NO	NO	NO	NO	NO
122	87	88	38	100	20	132	82	79	28	99	19	123	88	94	33	98	18	NO	104	125	90	11	NO	NO	NO	NO	NO	NO
122	89	95	31	99	21	118	88	90	25	99	20	105	84	88	31	98	19	NO	99	110	95	6.5	NO	NO	NO	NO	NO	NO
111	78	89	30	99	21	108	88	82	30	98	20	115	90	98	37	99	20	YES	128	142	100	7	YES	NO	YES	YES	YES	NO
130	90	94	28	99	20	120	80	88	33	98	19	125	80	84	34	99	19	NO	109	130	75	7	NO	NO	NO	NO	NO	NO
115	70	84	29	99	20	121	71	89	31	100	19	125	95	82	32	99	19	YES	127	138	98	7	YES	NO	NO	NO	NO	YES
119	85	80	32	99	21	120	80	84	30	99	20	115	75	80	31	99	19	NO	88	122	80	7.5	NO	NO	NO	NO	NO	NO
130	90	90	33	99	20	125	75	85	33	99	19	109	89	84	30	99	18	NO	95	111	90	6.5	NO	NO	NO	NO	YES	NO
109	75	82	30	99	19	100	80	74	31	99	18	124	84	86	26	99	18	NO	92	133	94	10.5	NO	NO	NO	NO	NO	NO
133	80	100	36	100	21	130	90	94	29	99	20	133	83	75	30	99	20	YES	106	149	100	7.5	NO	NO	NO	NO	NO	NO
129	98	95	34	100	19	125	85	89	28	99	19	129	90	82	32	99	19	YES	119	138	99	7.2	YES	NO	YES	YES	YES	NO
122	82	90	31	100	20	120	90	88	30	99	19	120	85	85	35	99	19	NO	99	110	90	7	NO	NO	NO	NO	NO	NO
105	70	88	33	100	20	111	70	90	33	98	19	115	76	80	31	99	19	NO	93	123	73	7	NO	NO	NO	NO	NO	NO
122	86	92	39	100	19	110	75	85	33	98	18	117	88	75	30	99	18	NO	88	129	87	11	NO	NO	NO	NO	NO	NO
125	85	95	33	98	20	120	90	93	35	98	19	111	82	88	27	99	18	NO	106	142	92	7.5	NO	NO	NO	NO	NO	YES
121	72	100	32	99	19	125	85	100	37	98	19	120	90	74	29	99	19	NO	102	133	88	7	NO	NO	NO	NO	NO	NO
130	90	89	36	99	20	129	89	90	30	99	19	130	88	88	33	99	18	NO	111	140	100	11	NO	NO	NO	NO	NO	YES
110	80	84	33	99	19	105	80	84	31	98	18	122	88	88	35	99	18	YES	120	132	80	7	NO	NO	NO	NO	NO	NO

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Govt. Rajaji Hospital,
Madurai.20. Dated: .08.2012

Institutional Review Board / Independent Ethics Committee.
Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,
Dean, Madurai Medical College & 2521021 (Secy)
Govt Rajaji Hospital, Madurai 625020.
Convenor
grhethicssecy @gmail.com.

Sub: Establishment-Govt. Rajaji Hospital, aMadurai-20-
Ethics committee-Meeting Agenda-communicated-regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00Pm on 28.06.2012 at the Dean Chamber, Govt. Rajaji Hospital, Madurai. The following members of the committee have been attended the meeting.

---

1. Dr.N.Vijayasankaran,M.ch(Uro.) 094-430-58793 0452-2584397	Sr.Consultant Urologist Madurai Kidney Centre, Sivagangai Road,Madurai	Chairman
2. Dr.P.K. Muthu Kumarasamy, M.D., 9843050911	Professor & H.O.D of Medical, Oncology(Retired)	Member Secretary
3. Dr.T.Meena,MD 094-437-74875	Professor of Physiology, Madurai Medical College	Member
4. Dr. S. Thamilarasi, M.D (Pharmacol)	Professor of pharmacology	
5.Dr.Moses K.Daniel MD(Gen.Medicine) 098-421-56066	Professor of Medicine Madurai Medical College	Member
6.Dr.M.Gobinath,MS(Gen.Surgery)	Professor of Surgery Madurai Medical College	Member
7.Dr.S. Dilshadh, MD(O&G) 9894053516	Professor of OP&Gyn Madurai Medical College	Member
8.Dr.S.Vadivel Murugan., M.D, 097-871-50040	Professor of Medicine Madurai Medical College	Member
9.Shri.M.Sridher,B.sc.B.L. 099-949-07400	Advocate, 2, Deputy collectors colony 4th street KK Nagar, Madurai-20.	Member
10.Shri.O.B.D.Bharat,B.sc., 094-437-14162	Businessman Plot No.588, K.K.Nagar,Madurai.20.	Member
11.Shri. S.sivakumar,M.A(Social) Mphil 093-444-84990	Sociologist, Plot No.51 F.F, K.K Nagar, Madurai.	Member

Following Projects were approved by the committee

21/8/12

Sl. No	Name of P.G.	Course	Name of the Project	Remarks
1.	Dr. Safeeba Burveen. H	M.D Anaesth	Endotracheal tube cuff air vs. alkalinized lidocaine in reducing tracheal morbidity.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance.

She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.

4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

13.8.12

DEAN I/c.

To
All the above members and Head of the Departments concerned.
All the Applicants.

21/12/12

DIRECTOR
INSTITUTE OF ANAESTHESIOLOGY
Madurai Medical College &
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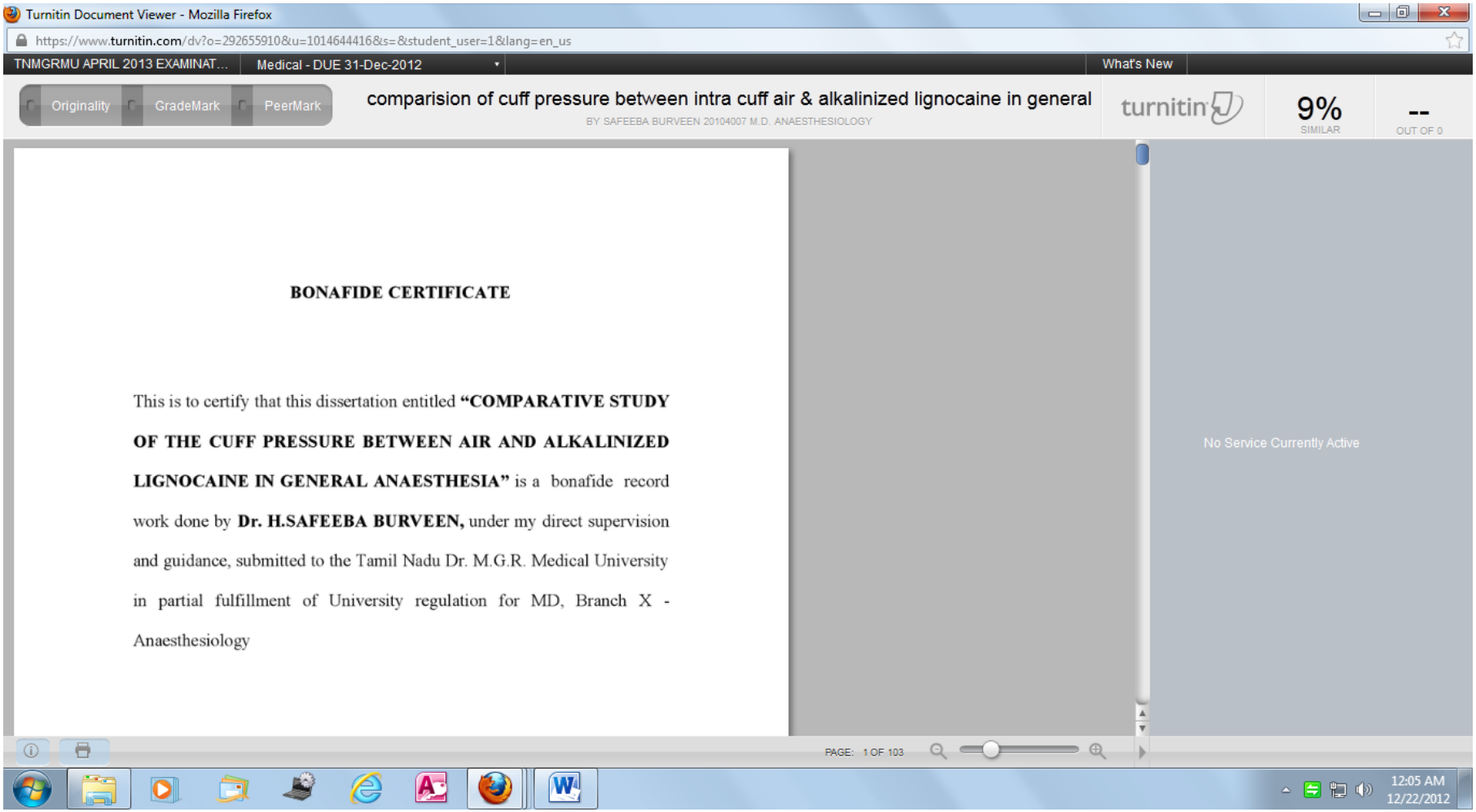
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